The Journal of Thoracic Surgery

VOL. 22 NOVEMBER, 1951 NO. 5

Original Communications

THE PROGNOSIS OF RESIDUAL BRONCHIECTASIS AFTER INCOMPLETE RESECTION

GEORGE P. ROSEMOND, M.D., W. EMORY BURNETT, M.D., AND JOAN HUMPHREY-LONG, M.D., (BY INVITATION)

PHILADELPHIA, PA.

ONE hundred ninety-four patients with bronchiectasis had pulmonary resections in Temple University Hospital from 1936 through 1949. We have been able to follow adequately 159 of these one to fifteen years. The response of the patient to surgery is pretty well established within six to twelve months after surgery, so we believe this follow-up study conducted by Dr. Long, who specializes in internal medicine and therefore took no part in the operative procedure, is unbiased and accurate, and has prognostic significance.

Since there is general agreement that bronchiectasis does not markedly influence longevity although it does impair health, we operate in an effort to relieve annoying symptoms and to make living more pleasant. The most important, constant, and annoying symptom of bronchiectasis is cough with expectoration. To justify surgery completely we should be able to offer to the patient some measure of assurance that this symptom will be eliminated with little or no risk to life and without producing or exaggerating dyspnea. The low mortality of lobectomy is well established.

This paper concerns itself with our successes, as well as our failures, in respect to cough with expectoration and, to a lesser degree, dyspnea.

TABLE I. NO COUGH—118 PATIENTS

A. All disease removed (except in 14 patients)	
B. Presence or absence of dyspnea depends on pulmonary function	
1. Adequate function	
No dyspnea	71 Patients
2. Markedly impaired function	
Dyspnea	47 Patients

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

From Temple University Hospital.

TABLE II. COUGH STILL PRESENT—41 PATIENTS

A. All have residual disease
Improved—36
No better— 5
B. Dyspnea is not correlated with function

TABLE III. RESIDUAL DISEASE—55 PATIENTS

	PATIENTS	PERCENTAGE
No cough or symptoms	14	25.5
Less cough, but still present	36	65.5
Increasing cough	5	9.0
Total	55	100.0

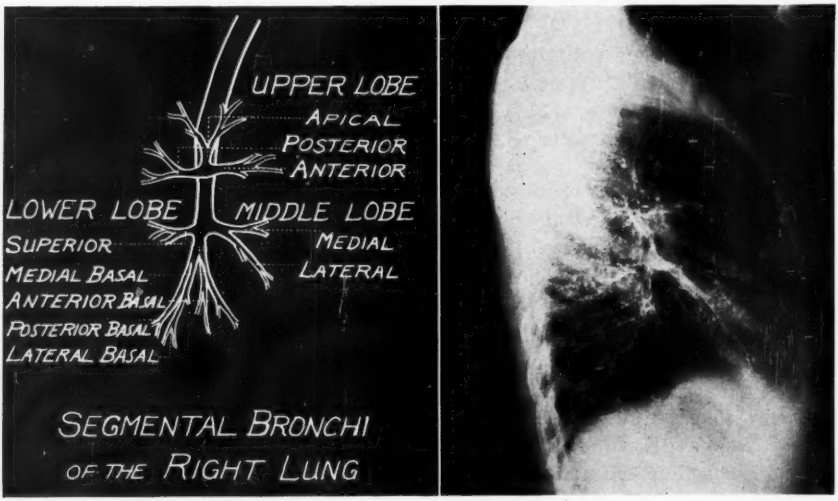


Fig. 1.—Lateral view of bronchogram of normal right lung demonstrating each individual segmental bronchus.

Of the 159 patients adequately followed, 118 are free of cough. Only fourteen of these have any residual disease. In the patients with unilateral bronchiectasis, pulmonary ventilation studies were essentially unchanged by surgery. Many of the patients complained less about dyspnea when infection and sputum were eliminated.

Forty-one patients still have some cough with expectoration, although thirty-six of these are improved. All of these patients have residual disease, and cough

TABLE IV. PATIENTS WITH RESIDUAL DISEASE

	PATIENTS	PERCENTAGE
Bilateral bronchiectasis	48	87
Unilateral bronchiectasis	7	13
Total	55	100

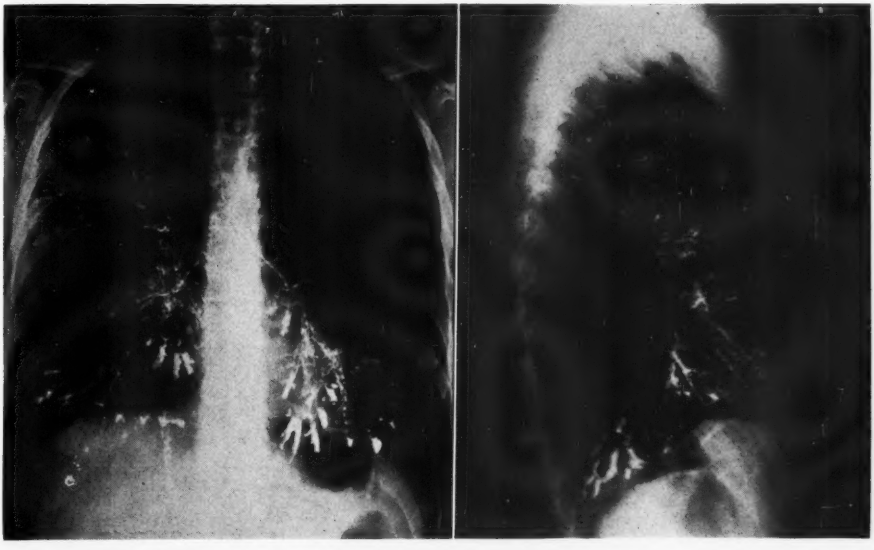


Fig. 2*A*.—(L.C.) Bronchogram of right lung preceding left lower lobe lobectomy as part of bilateral stage procedure.

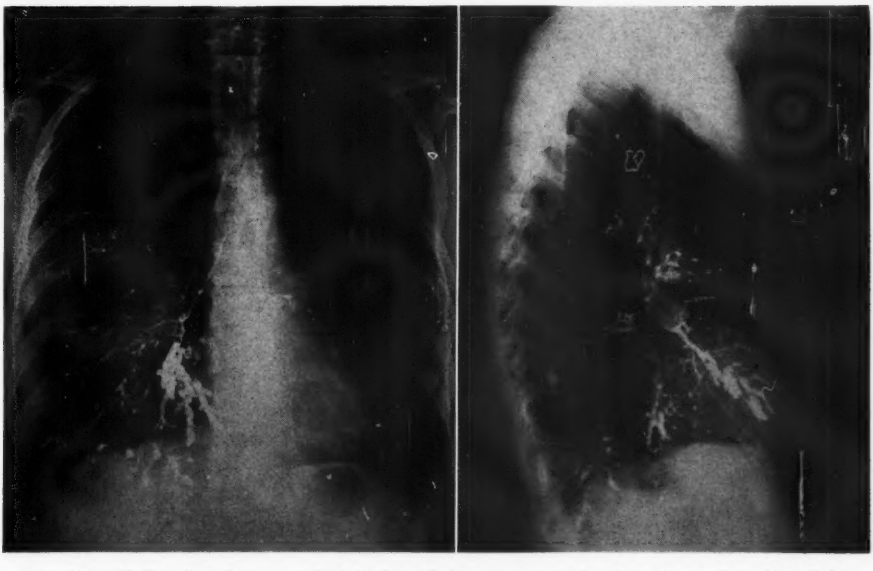


Fig. 2*B*.—(L.C.) Bronchogram of right lung between stages showing disease in middle lobe missed by original bronchogram.

is, in most instances, proportionate to the degree of residual bronchiectasis. Also, with residual disease, dyspnea is not correlated with ventilation, subjective complaints of dyspnea being out of proportion to the reduction in ventilation.

Of the fifty-five patients with residual disease, 14 (25.5 per cent) have no cough or symptoms of any kind at the time this study was compiled, although, of course, we do not know how long this will last.

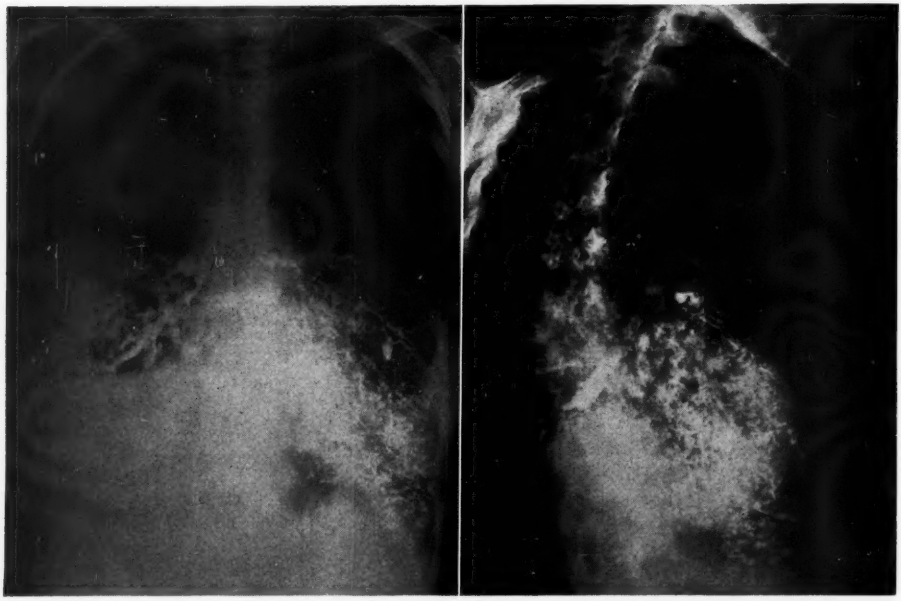


Fig. 3.—(E.W.) This is an example of a too vigorous attempt at saving normal lung. Removal of one more segment on each side would have eliminated cough, and probably would have made little difference in ventilation.

Of the fifty-five patients with residual disease, forty-eight had bilateral bronchiectasis, and only seven had unilateral bronchiectasis. Many of these patients have mild disease producing mild symptoms, but it is also likely that we have tended to be overly conscientious in conserving lung structure when bilateral resection is anticipated.

Thirty-nine (71 per cent) of this group of fifty-five patients had known minimal disease on the opposite side and further resection is being considered.

TABLE V. CAUSES FOR RESIDUAL DISEASE

	PATIENTS	PERCENTAGE
Known minimal disease on opposite side	39	71.0
Judgment at time of surgery	3	5.5
Faulty evaluation	13	23.5
Total	55	100.0

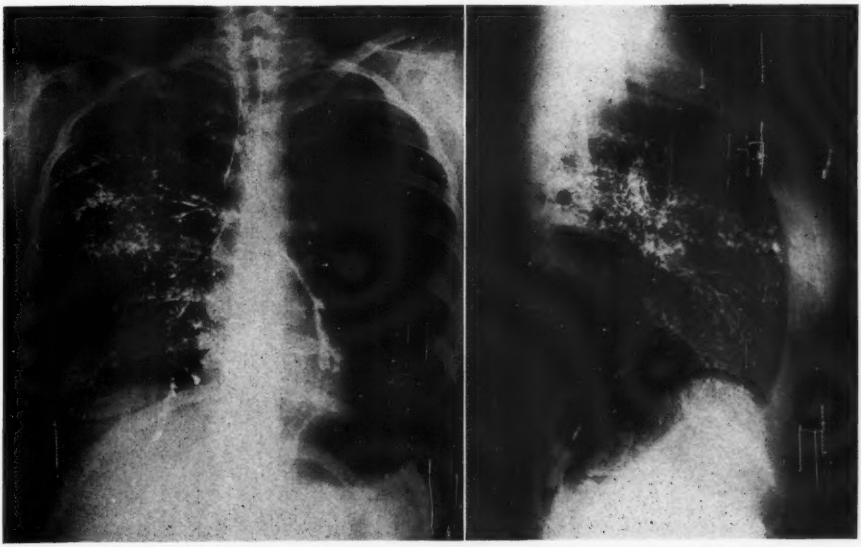


Fig. 4*A*.—(L.W.) Demonstration of poor filling of superior segment of the right lower lobe on bronchography. Segment was allowed to remain at surgery.

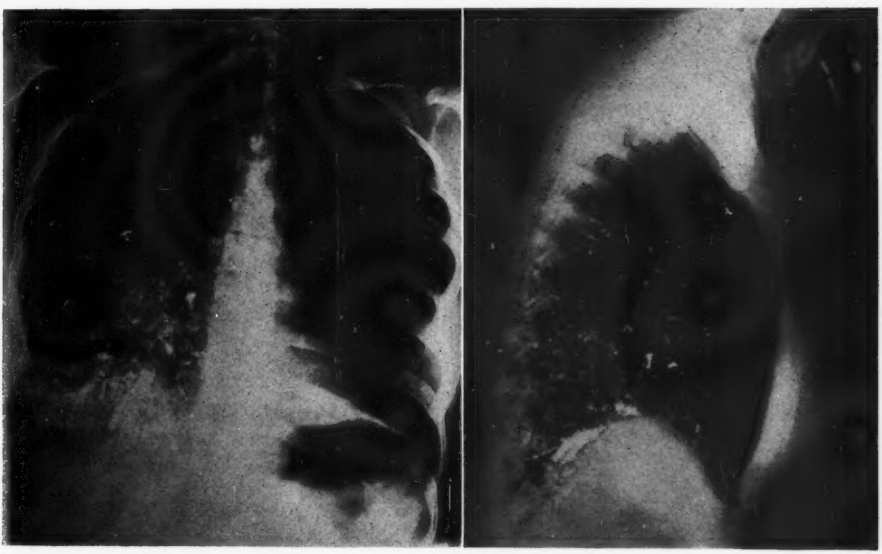


Fig. 4*B*.—(L.W.) Residual disease in displaced superior segment after basal segments were removed. Symptoms are in proportion to residual disease.

Three (5.5 per cent) have disease remaining because it was considered better judgment to leave some less severely diseased lung than to chance removing too much. This judgment we now believe was faulty. It is suspected that patients who are operated on bilaterally do tend to have reduced ventilation, although our series of this type is so small that we hesitate to be dogmatic about this.

Thirteen (23.5 per cent) have residual disease due to faulty evaluation.

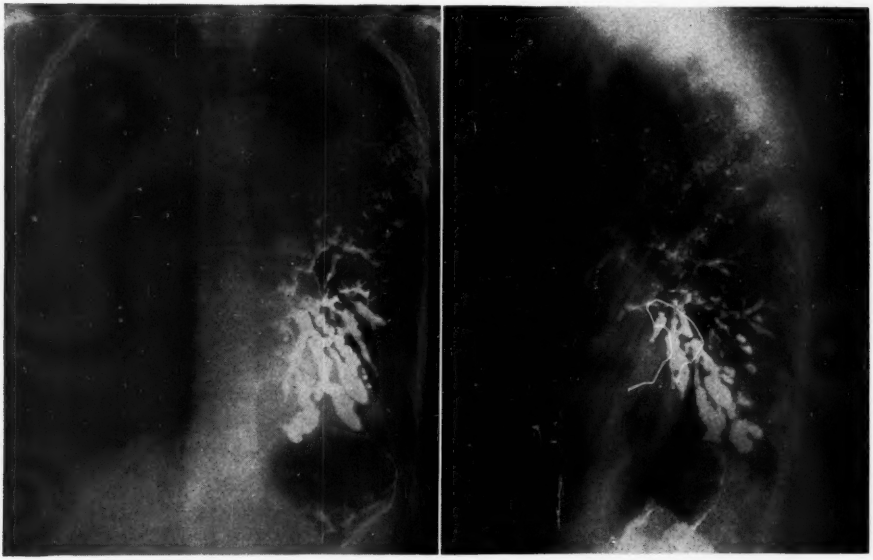


Fig. 5A.—(F.H.) Bronchogram of left lung showing saccular bronchiectasis.

These factors may contribute to incomplete resection. Since accurate surgery depends so entirely on bronchography, a faulty bronchogram should not be accepted. Bronchography should be so clear that each segmental bronchus can be identified and alveoli demonstrated (Fig. 1).

Bronchography should be done with the lung as free of secretions as possible. The preparations used will not displace secretions, and iodized oil is so slowly absorbed that after one incomplete mapping with lipiodol, further evaluation is difficult. The water-soluble preparations now available have not proved themselves as yet. The ones we have used have been so viscid that they

TABLE VI. REASONS USUALLY CONTRIBUTING TO RESIDUAL DISEASE

1. Faulty bronchogram
2. Limitations of method of bronchography
3. Attempted evaluation at operation
4. Fear of removing "too much lung"
5. Improper anatomical evaluation of segments
6. Surgical technique

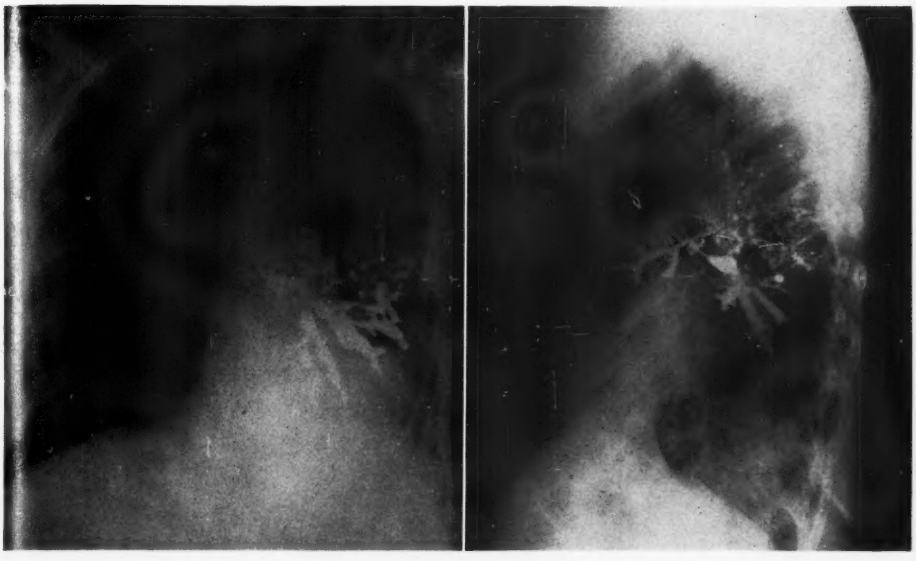


Fig. 5*B*.—(F.H.) Repeat bronchogram of left lung after palliative removal of left lower lobe. As is obvious, considerable disease remains.

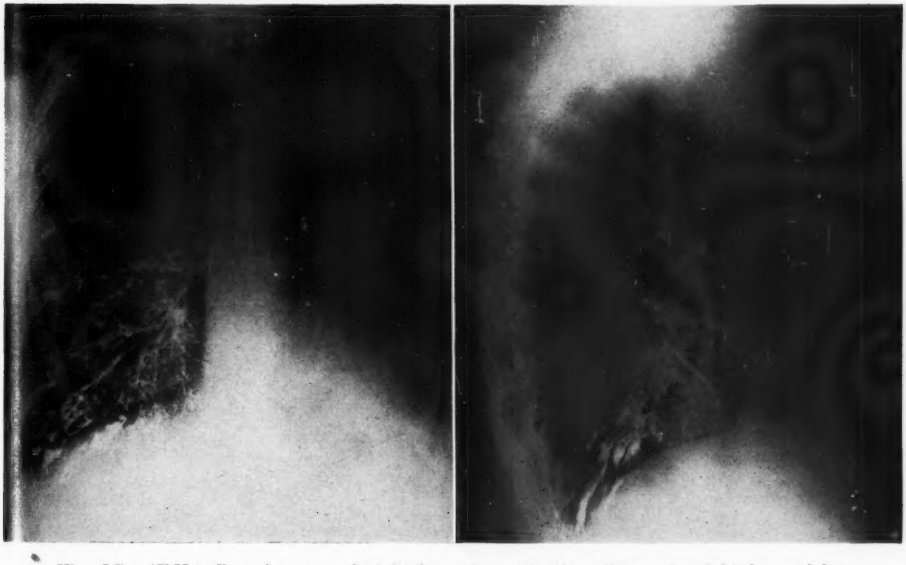


Fig. 5*C*.—(F.H.) Bronchogram of right lung demonstrating disease in right lower lobe.

Fig. 6*A*.—(C.W.) Bronchogram of left lung showing extensive disease.

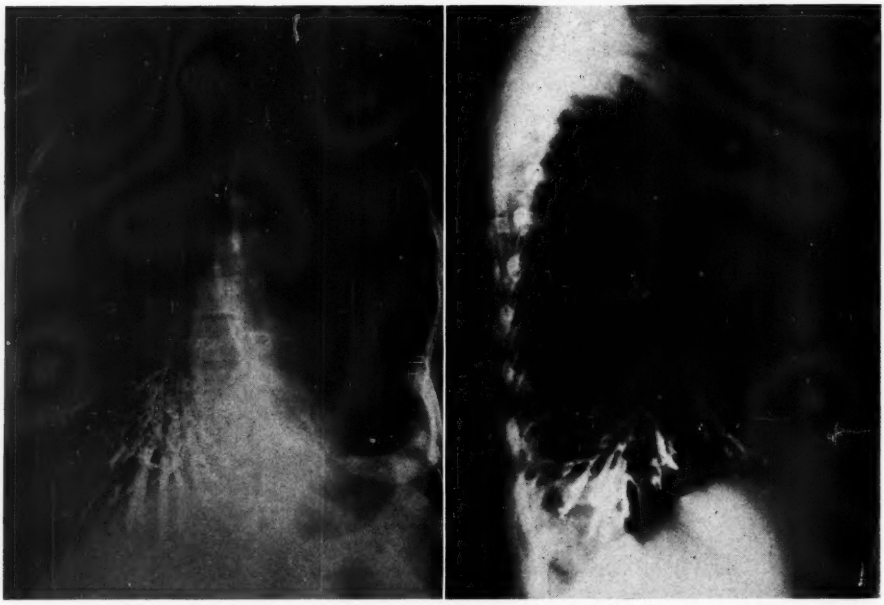


Fig. 6*B*.—(C.W.) Bronchogram of right lung after surgery on left side. This side shows much less involvement, but the patient was not relieved of symptoms until the disease was eradicated from this less severely affected right side.

do not fill the alveoli well. The rapid absorption of water-soluble preparations, which would allow repeated bronchography as often as necessary to determine the extent of disease, gives us hope that an improved preparation of this nature may be developed soon. We have learned to repeat lipiodol mapping in bilateral cases between stages to avoid being fooled by secretions (Fig. 2 *A, B*).

Fig. 3 is an example of shaving too close at operation. One more segment would have eliminated cough and would not have impaired ventilation.

Fig. 4*A* shows poor filling of the superior segment of the right lower lobe. This segment was not removed at operation. Fig. 4*B* shows residual disease in the displaced superior segment. The patient's symptoms are in proportion to the amount of residual disease.

Fig. 5*A, B,* and *C* demonstrates a case of saccular bronchiectasis in which palliative resection was done. There is residual disease present because we considered it poor judgment at the time to remove so much lung on one side in the presence of bilateral disease. Evaluation of pulmonary ventilation before and after resection has led us to believe that this reasoning was erroneous. This patient, incidentally, has no symptoms.

The patient whose films are shown in Fig. 6*A* and *B* had very little change in symptoms until the apparently less involved lobe was resected. She is now symptom free.

Since it is impossible to evaluate the extent of bronchiectasis at operation, preoperative bronchography must be accurately carried out. If external spirometry and bronchospirometry are done preoperatively, one has a fairly good means of evaluating the function which can be expected postoperatively. Removal of all bronchiectatic lung is the only way to insure freedom from cough with expectoration.

DISCUSSION

DR. J. H. WALKER, Brookline, Mass.—I agree with the speaker that the total removal of the diseased tissue is necessary in the treatment of bronchiectasis if one is to obtain a good result; but also, to qualify that statement, one should conserve all normal pulmonary segments at the same time in order to achieve the maximum benefit. We have had experience with the surgical treatment of 150 odd cases of bilateral bronchiectasis in the Overholt Thoracic Clinic, and it has been necessary to do bilateral surgery in only one-third of the cases. We have recently brought back a few of the cases for pulmonary function studies to evaluate effects of bilateral segmental resection in severe bronchiectatic cases.

(Slide.) These are four patients that have had extensive bilateral surgery; three of these I am sure could be classified as total respiratory cripples prior to surgery. They had severe cough with sputum averaging from one-half cup to two cups per day. They are now free of cough and purulent sputum. You will note in the first column the total number of segments that were removed varied from seven to eleven. The girl who had eleven segments removed had segments removed from all five lobes. The percentage of predicted maximum ventilation postoperatively ranged from 48 to 81 per cent for total pulmonary function. This is a nice surprise, and is better than we had anticipated with bilateral excisional therapy.

(Slide.) This is a breakdown of the number of segments removed from each lung and also the differential bronchospirometry. There is a fairly close correlation here between the number of diseased segments removed, the number of normal segments left behind, and the proportion of ventilation carried by each lung. This emphasizes not only the necessity of removing diseased segments, but also the preservation of all normal pulmonary segments.

DR. J. GORDON SCANNELL, Boston, Mass.—As a footnote to the paper by Dr. Rosemond, I would like to present the by-product of a recent study we have conducted at Massachusetts General Hospital on what happens to patients a long time after the diagnosis of bronchiectasis has been made. Looking back in our records we find that in 1939-1940, there were some eighty-seven patients admitted in whom a diagnosis of bronchiectasis was made. Of this number forty-three were operated on, but there was a small group of nineteen in whom the diagnosis was made and in whom the anatomic indications for resection were present, but in whom, for one reason or another, operation was not carried out. In the past month I have been able to trace seventeen of these; of the seventeen, eleven are in excellent health. Four—and this was rather surprising to me—died of fatal massive pulmonary hemorrhage; and one died of general decay which was obvious when he was in the hospital. It is interesting that recently none of the patients have died of sepsis, and it is obvious also that penicillin had a tremendous modifying effect in the past few years.

DR. BURNETT (Closing).—In closing this discussion I simply want to remind us all of the fact we have recognized so often; that impressions gained are frequently dispelled by a little analysis. When this analysis was made, I was surprised at the number of patients in our group who had had residual disease. I would not have believed that from the impression gained as we went along. Second, I would like to emphasize the value of palliative resection in extensive disease. We have not tackled any patients with five lobes involved, as Dr. Walker and Dr. Overholt have done; we have not had quite that much courage, but segmental resection certainly makes this possible. We have, however, utilized segmental resection in a high percentage of these patients.

One of the most extreme examples of palliative resection was one young lady from South Africa who had five lobes involved, who had very advanced disease in the left lower lobe, and moderate to advanced in others. After removal of the left lower lobe and lingula her symptoms were decreased by about 90 per cent, which she felt was quite worth while, and we agreed. Moderate involvement on the opposite side to that which is symptogenic and saccular certainly warrants conservatism on the opposite side, as Dr. Rosemond has demonstrated. We believe it may be well to observe those patients and see if symptoms warrant further attack. As you saw, some fourteen of those patients, although having residual disease, had no symptoms whatever, and the others were markedly improved, with the exception of five.

I want to thank Dr. Walker and Dr. Scannell for their discussion, and to say that we will gather great impetus from their suggestions.

RADICAL PNEUMONECTOMY

WILLIAM G. CAHAN, M.D. (BY INVITATION), WILLIAM L. WATSON, M.D., AND JOHN L. POOL, M.D.
NEW YORK, N. Y.

THE purpose of this report is to submit data showing that a lung can be removed in a block dissection together with most of its regional hilar and mediastinal lymph nodes.

The technical steps and the clinical implication of the procedure to be discussed are based upon an experience of thirty-nine such operations successfully performed on the Thoracic Service of Memorial Hospital.

DEFINITION

The technique of lung removal for primary carcinoma of the lung has been described many times since the first successful one-stage pneumonectomy in 1933.[6] However, although rapid progress has been made in the management of the complete physiologic and anatomic problems inherent in this procedure, there remain several confusing and controversial features which require clarification.

In the literature the term, "pneumonectomy," has been employed either alone or with the adjectives, "simple," "total," or "dissection." These terms have been used interchangeably and, as a consequence, there is some confusion as to whether an actual difference exists. When some of the hilar or mediastinal lymph nodes have been included in the procedure, these have been alluded to for the most part in indefinite terms as to their location or whether they were individually excised or taken en bloc with the lung.[12] For over two years we have been distinguishing the removal of a lung alone—or simple pneumonectomy—from cases in which mediastinal node dissection is included. During this time we have been impressed, in concurrence with the views expressed by Allison[1] last year, with the need for an inclusive term for the more radical procedure. It is suggested, therefore, that these operations for lung cancer be defined as follows:

A *simple* pneumonectomy is the excision of the lung alone.

A *radical* pneumonectomy is the excision of the lung in continuity with its regional lymph nodes located in the hilar and mediastinal areas.

When adjacent structures are included in the procedure they may be added as follows: e.g., a "simple pneumonectomy with resection of ribs 3, 4, and 5"; or a "radical pneumonectomy with a partial pericardiectomy."

The desirability of removing a malignant tumor en bloc with its regional lymphatic drainage area is an established principle of cancer surgery when certain circumstances exist:

From the Thoracic Surgical Service, Memorial Hospital.
Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

1. No distant metastases are discernible;
2. No other form of therapy has any real value;
3. The morbidity and mortality rate of the surgical procedure required remains reasonably low;
4. From experience, a high index of probability exists for present or future involvement by cancer of the lymphatic pathways to be excised;
5. If these lymphatic areas are not resected at the time of the original procedure, they would be hidden within the body so that their future involvement by metastases would escape early detection.

These are principles that have been applied to the treatment of cancers arising in the colon, stomach, and breast.

It is known that the hilar and mediastinal lymph nodes can become involved by metastases from a primary carcinoma of the lung. Ochsner[8] reported in 3,047 collected cases of carcinoma of the lung that these nodes were involved in 72.2 per cent. His relative distribution of lymph node metastases in 1,298 collected cases showed that 69.7 per cent were in the tracheobronchial group, 20.7 per cent in the abdominal, 17.4 per cent in the cervical, and 4.2 per cent in the supraclavicular areas.

Figures from Koletsky's[7] series of 100 autopsies support the fact that regional lymph nodes are indeed the chief site of early metastasis for most lung carcinomas.

In consideration, then, of the traditional treatment of cancer for other sites and the assembly of pathologic facts as listed above, it would seem that any practice of plucking out a lymph node here and there "like raisins from a bun" in these areas should be discouraged if a method can be devised to excise them in continuity with the primary tumor. It is felt that by a relatively detailed anatomic and physiologic description of pulmonary lymphatic drainage, the possibility and techniques of such a block dissection will become apparent and appear reasonable.

LYMPHATIC SYSTEM OF THE LUNG

No article on lymph node drainage should begin without a tribute to the excellent studies of Rouviere[10] and it is strongly recommended that his original work be consulted for a more detailed and thorough account than that which follows and which was derived liberally from it. Rouviere points out that even as there are different anatomic features to the contents of the right and left thoracic cavities, so are there variations in the lymph node pattern and drainage on both sides. He defines several major divisions of node-bearing areas; mediastinal, peritracheobronchial, and intrapulmonary.

MEDIASTINAL LYMPH NODES

The anterior mediastinal, or prevascular nodes, are placed in the superior portion of the mediastinum in front of the large blood vessels, which are connected with the heart. On the right side, the chain is placed in front of the superior vena cava and the right innominate vein in a pre-venous location with

one prominent node at the junction of both innominate veins. Lymph nodes about the right phrenic nerve and inferior vena cava also drain into this pathway, terminating at the right jugulo-subclavian junction or the right lymphatic duct.

The left anterior mediastinal chain is pre-arterial, or, more exactly, pre-aortico-carotid. It commences in a node of good size placed just in front of the ligamentum arteriosum, which rests on the left pulmonary artery. The pre-aortico-carotid chain (left anterior mediastinal chain) ascends between the phrenic and the vagus nerves, following chiefly the course of the latter nerve. The nodes of this chain are placed from below upward, at first in front of and on the superior border of the aortic arch, then above this vessel on the anterolateral surface of the common carotid artery. Quite often a few nodes are placed somewhat medially along or a little in front of the right lateral border of the thymus gland. The left lymphatic mediastinal path terminates in the thoracic duct or directly into the jugulo-subclavian venous junction.

The transverse anterior mediastinal chain nodes are placed along the superior and inferior borders of the left innominate vein and may form an anastomosis between the right and left mediastinal pathways, as well as between the right and left paratracheal chain of nodes.

The *posterior* mediastinal nodes are juxta-esophageal. Normally 2 to 5 nodes are placed along the lateral borders of the esophagus at the level or somewhat below the inferior pulmonary vein and occasionally between the esophagus and the aorta. The greater number of the efferent vessels from these pour their contents into the subcarinal (inter-tracheobronchial) nodes while the rest empty into the thoracic duct.

PERITRACHEOBRONCHIAL NODES

These include the paratracheal nodes, subcarinal (or nodes of the bifurcation), and the nodes of the pulmonary roots. Two principle chains of nodes are present: the right and the left lateral or paratracheal chains and a few inconstant retrotracheal nodes.

a. *Right.*—The paratracheal nodes are placed in a niche, the boundary of which is as follows: in the front, the superior vena cava and the right innominate vein behind, the right anterolateral surface of the trachea; medially, the arch of the aorta and the innominate artery; above, the right subclavian arteries surrounded by the right recurrent nerve; below, the arch of the vena azygos; laterally, the niche is closed by that part of the mediastinal pleura which extends from the superior vena cava and the right innominate vein to the right lateral border of the trachea, and which is skirted by the vagus nerve.

The right paratracheal chain consists of three to six nodes which may be arranged in a single file, although most of these are in groups of two to three in juxtaposition. The chain is not truly vertical but bends somewhat posteriorly. The highest node is at the same time the most laterally placed and the most posterior; and is quite often remarkable for its size, particularly when it receives a large lymphatic trunk coming from the nodes at the bifurca-

tion. It rests immediately below the right subclavian artery but may insinuate itself partly behind this vessel. Above this node arise the nodes of the right recurrent chain. Generally, the lowest node of this right paratracheal chain is the largest and is always found at the arch of the vena azygos, which in the majority of cases covers its lower pole, or actually the node may project over this vein. It has been called the "node of the arch of the vena azygos."

Afferent and efferent lymphatics in this group form a very important ascending right paratracheal lymphatic path, which is a continuation of the vessels which issue from the right lung, the right supra- and retrobronchial nodes, and from the nodes of the bifurcation. This path also receives directly some lymphatic vessels from the trachea, the esophagus, and the thymus gland. It terminates above either in the right lymphatic duct or in a separate entrance into the jugulo-subclavian angle.

b. *Left.*—The *left* lateral paratracheal chain conists of 4 to 5 nodes placed along the anterior and medial aspects of the vertical segment of the left recurrent nerve, occupying a position along the recurrent nerve lateral to the left posterolateral border of the trachea, medial to the pleura and medial and posterior to the arch of the aorta and the left subclavian artery. They ascend in the left paratracheal lymphatic path.

The retrotracheal nodes are placed behind the inferior part of the trachea on the path of the lymphatic vessels which unite the subcarinal nodes to the right paratracheal chain. These are present in about one-eighth of the cases. Generally there is but one node placed a little above the tracheobronchial angle near the right border of the posterior surface of the trachea along the esophagus and the vagus nerve.

SUBCARINAL NODES (NODES OF THE TRACHEAL BIFURCATION)

This group occupies the interval between the trachea formed by the divisions of its two bronchi and above the inferior pulmonary veins. There are usually 3 to 5 nodes which are fused in such a way as to have a mammiliform shape indicating an incomplete division of this node into smaller elements. The total number of elements in it varies. These nodes are in contact with the inferior border of the trachea and the suprajacent bronchus. They even extend somewhat in front of the tracheal spur, and there is a bond of union between the subcarinal nodes and the inferior extremities of the paratracheal chain. They are in relation with the pericardium in front and with the esophagus, vena azygos, and aorta behind. The efferent lymphatics from these nodes usually proceed to the right paratracheal chain (rarely to the left paratracheal chain), and reach their destination and pass either in front of or behind the tracheobronchial tree.

NODES OF THE PULMONARY ROOTS

A group of nodes is placed between the elements which make up the pulmonary root. They are listed as anterior, posterior, superior, and inferior, according to their position and relation to the respective bronchus. They are

present if anterior in pre-venous, pre-arterial, and pre-bronchial locations. The posterior nodes are found more frequently on the right side than on the left. The superior nodes, about 1 to 3 in number, are found most often on the left side, lie along the superior border of the bronchus, and behind the left pulmonary artery. This chain is constant and continuous above with the lateral tracheal, left recurrent chain of nodes by means of a lymphatic vessel which passes below the arch of the aorta and the ligamentum arteriosum (Fig. 1). There is a node of the loop of the left recurrent nerve at this level. Arnstein attributes certain cases of paralysis of the left recurrent nerve to tuberculosis of these lymph nodes. This might also be true when metastases are present, which burst the nodal capsule and invade the nerve secondarily.

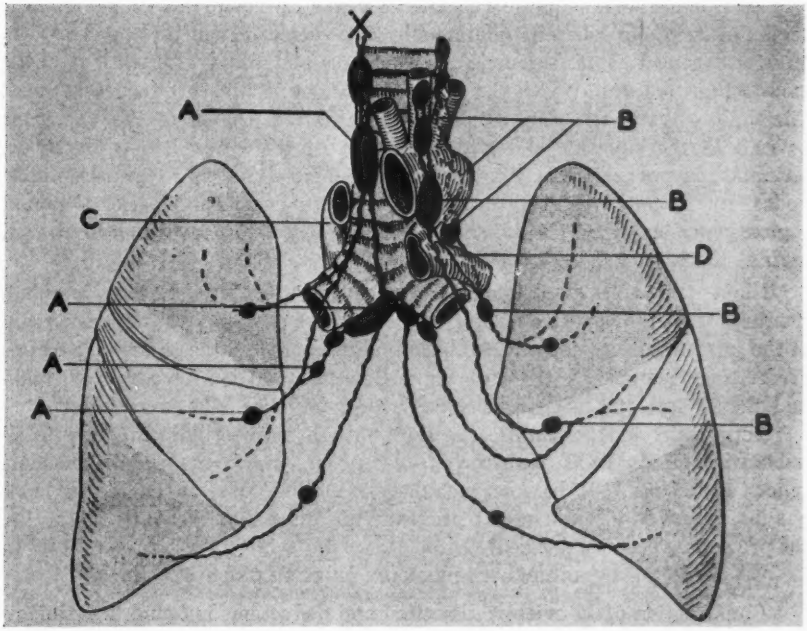


Fig. 1.—Diagram illustrating lymphatic drainage areas of the lungs. These are divided into superior, middle, and inferior regions. The right lung drains entirely up the right lymphatic pathway. The lower two segments of the left lung drain predominantly to the subcarinal nodes and then up the *right* lymphatic pathway. Occasionally, they may also travel up the left lymphatic pathway, joining the drainage from the superior section of the left lung. (Note that the sections are not confined by major fissures.) Drinker after Rouviere.

The *pre-esophageal* nodes of the inferior pulmonary ligament extend from just below the bronchus to the diaphragm, and are found more often on the left than on the right side (the latter in only 20 per cent of the cases according to Rouviere). These drain to the subcarinal nodes.

INTRAPULMONARY NODES

Nodes are placed along the broncho-arterial trees of each lobe, and it is important to note that the principal node draining one lobe group may rest against the bronchus of an adjacent lobe.

Each lung has three principal lymphatic regions—superior, middle, and inferior (Fig. 1). These are not limited by the usual anatomic boundaries or major fissures.

Right Lung.—The superior lymphatic region is represented by the anteromedial portion of its upper lobe. Its collecting vessels are tributaries solely of the right paratracheal nodes, particularly of the node of the arch of the vena azygos. The middle lymphatic region which includes the posterolateral region of the upper lobe, all the middle lobe, and the superior part of the lower lobe conveys lymph to both the right lateral tracheal and subcarinal nodes. The inferior lymphatic region comprises the inferior region of the lower lobe; the lymph from this region is poured entirely into the subcarinal nodes.

Left Lung.—The superior lymphatic region is comprised of the superior region of its upper lobe, the lymph collecting vessels of which connect with the left paratracheal chain, the node of the ductus arteriosus with the left anteromediastinal nodes and with the node of the loop of the left recurrent nerve. The middle lymphatic region includes the inferior region of the upper lobe as well as the superior and middle regions of the lower lobe. The vessels terminate in part superiorly in the anterior mediastinal and left paratracheal nodes, and in part inferiorly in the subcarinal nodes. The inferior lymphatic region represented by the inferior part of the lower lobe drains to the subcarinal nodes.

Since the subcarinal nodes normally convey their efferent lymphatic trunks to the right paratracheal group, it follows that only the superior region of the left lung in its entirety, and the middle region of the same lung in part, divert their lymph into the anterior mediastinal and left paratracheal nodes, and thence into the left jugulo-subclavian confluence. The remainder of the left lung, part of the middle region, and the entire right lung are finally tributaries of its right paratracheal chain and the right jugulo-subclavian venous confluence. It becomes possible to foretell to some degree by which lymphoid echelon a lesion might progress, the exact seat of which in the lung has previously been established.

LYMPHATIC DRAINAGE OF THE LUNG

Confirmation of Rouviere's dissection and deductions is found in Drinker's[5] report (Fig. 1). He cannulized separately the right lymphatic and the thoracic ducts in dogs. Carbon particles were then deposited carefully by bronchoscope in various segments of the lung and their reappearance in the lymph flow of the right or left cannulas noted.

As might be anticipated from Rouviere's work, when the particles were placed in any segment of the right lung or in the lower two-thirds of the left lung, the lymph emerging from the right cannula (x in Fig. 1) was colored by them, while that from the thoracic duct remained clear. Only when particles were deposited in the superior portion of the left upper lobe did they appear in the lymph issuing from the left cannula.

These experiments have found clinical confirmation. It has been noted that metastatic lymph nodes located in the right supraclavicular region just

behind the clavicular head of the sternocleidomastoid muscle can be traced to lung carcinomas having their origin in the right lung or in the middle and lower lymphatic regions of the left lung. In addition, the surgeon has a unique opportunity to observe at the time of exploratory thoracotomy, the intervening channels between a primary carcinoma and the highest apparently involved lymph node. This may be graphically demonstrated in the future even in unresectable cases when these areas are delineated by the use of dyes according to the technique of Weinberg and Greaney.[13]

Specimens removed by radical pneumonectomy with their lymph node groups intact and clearly marked so as to maintain their relative position will aid further in delineating the pattern of metastatic spread.

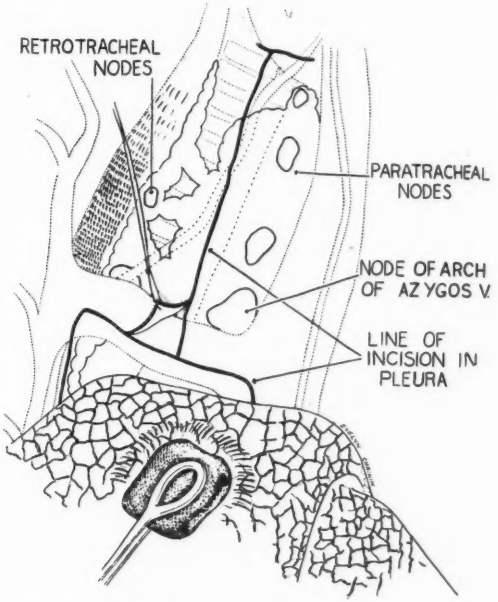


Fig. 2.—Right side view of paratracheal lymph nodes and superior mediastinal pleural incision.

Finally, a careful study of metastatic lymph node involvement from autopsy specimens of patients who have died of lung cancer has already provided, and will continue to provide a real aid in determining the flight of metastases. In this connection, Crafoord[4] reported in his early articles at least three autopsies on patients who had had what was probably a simple pneumonectomy that revealed residual metastatic lymph nodes located in the paratracheal regions.

To be sure, variations in the pathways described above occur. In particular, metastasis from the lower two-thirds of the left lung may travel directly up the left side instead of by the more usual contralateral spread.

In addition, with the choking of one node by metastasis, retrograde extension along pathways not ordinarily taken may occur. Skipping of lymph nodes by metastases has also been noted. By and large, however, metastases follow the echelons outlined above.

This anatomic and pathologic knowledge suggests that removal of the described mediastinal lymph pathways with the lung will improve end results. One may speculate, however, that primary lesions of the lower two-thirds of the left lung will have a poorer prognosis because contralateral lymph node metastases will preclude block dissection.

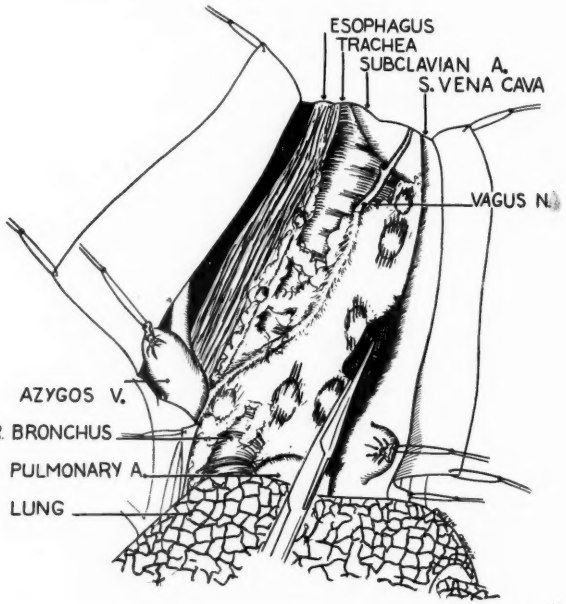


Fig. 3.—Right side: Pleural flaps elevated. Azygos vein divided. Dissection begun beneath superior vena cava.

TECHNIQUE

To approach either lung the patient is placed in a true lateral position and the chest entered through a long parascapular incision and the bed of the fifth rib. The pathologic setting is explored and, if a radical pneumonectomy seems feasible, and the patient's general condition is satisfactory, the procedure is carried out usually in the following sequence.

Right Side.—

1. *Incision* (Fig. 2): The superior mediastinal pleura is incised from the apex of the chest to the azygos vein arch just anterior to the vagus nerve. Superiorly, a short horizontal limb is used to facilitate exposure of the subclavian artery and right recurrent laryngeal nerve.

Inferiorly, a horizontal incision follows the azygos vein to the phrenic nerve and thence inferiorly along this nerve to the inferior pulmonary ligament. Posteriorly, the incision of pleura is just anterior to the azygos vein all the way to the diaphragm where it meets the anterior incision.

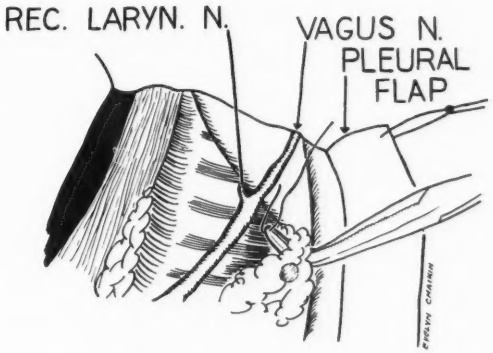


Fig. 4*A*.—Right side: Apex of dissection beneath right subclavian artery.

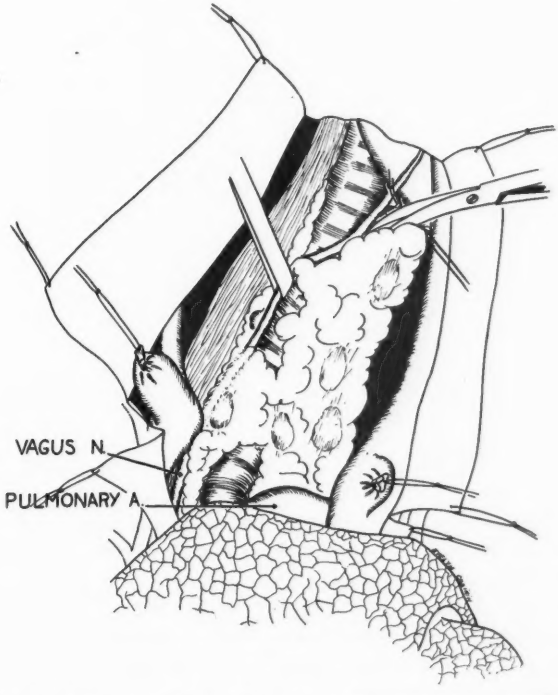


Fig. 4*B*.—Right side: Retraction of vagus nerve and dissection of paratracheal node-bearing tissue from it. Note clearing of retrocaval space.

In keeping with the tradition of starting a block dissection for cancer with the distantmost points of potential metastasis, the first section of the procedure progresses from the most peripheral nodes to the hilum.

2. *Paratracheal dissection* (Fig. 3): The anterior pleural flap is elevated to the anterior surface of the superior vena cava, phrenic nerve being displaced with it. The posterior flap is elevated to the vertebral bodies, leaving vagus nerve uncovered. These flaps are retracted by the weight of Halsted clamps fastened to sutures attached to their free edges.

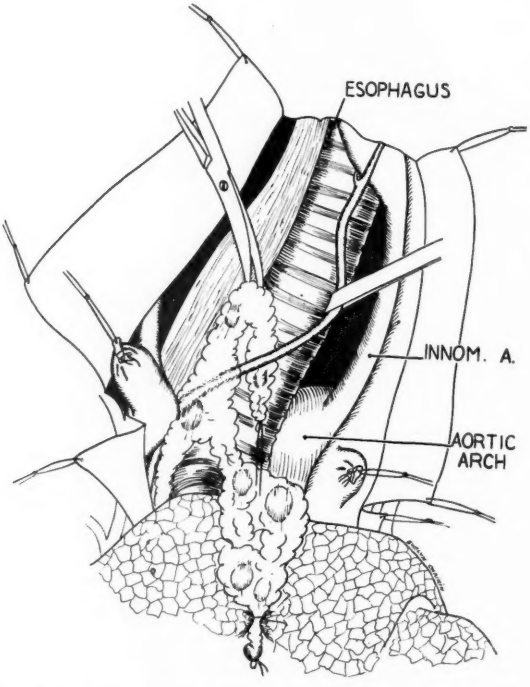


Fig. 5.—Right side: Retrotracheal lymph node dissection and vagus nerve elevated to pass this group beneath it. Note the most constant node at the tracheobronchial angle. Also the boundaries of the retrocaval space.

The azygos vein is transected between ties and ligatures, and the anterior limb lifted forward, thus also elevating the superior vena cava. The areolar tissue is cleared laterally off the latter vein, and its posterior surface freed.

The vagus nerve is mobilized, and superiorly its recurrent laryngeal branch is usually identified. (Fig. 4 [insert]).

The apex of the dissection is the inferior border of the subclavian artery where a tongue of areolar, node-bearing tissue is isolated and divided. Next (Fig. 4), attention is turned to the trachea and the fine connective tissue on its right lateral and anterior surfaces which is found to separate readily allowing the whole packet of tissue anterior to the trachea to be swept downward off

innominate artery and aortic arch to the level of the pulmonary artery. This can usually be done with division of only one or two small vessels. This packet of tissue represents the right paratracheal lymph node chain. To maintain the integrity of this block of lymphatics, the dissection must be carried immediately adjacent to the large vessels and the trachea.

The areolar tissue posterior to the trachea and on the esophagus (Fig. 5) is then freed from these structures, commencing superiorly, dissected downward, and passed under the vagus nerve which is elevated by a Penrose drain. If desired, this last structure can be sacrificed below the recurrent nerve origin.

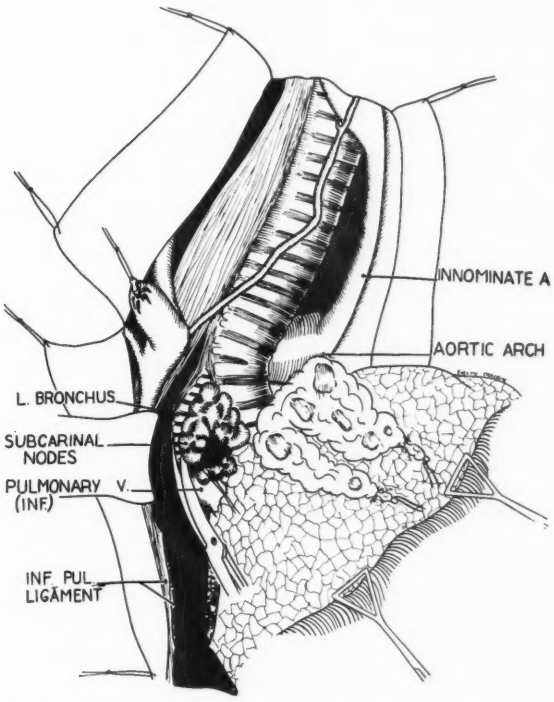


Fig. 6.—Right side: Pre-esophageal nodes have been dissected with the inferior pulmonary ligament. Traction on the lung and on the esophagus exposes the subcarinal lymph nodes. Dissection begun at contralateral bronchus.

Included in this dissection is the particularly important, constant, and large node of the azygos arch lying just above the pulmonary artery. It is mobilized as the dense connective tissue superior to the pulmonary artery and behind the superior vena cava is incised. This paratracheal node packet remains attached to the lung by peribronchial lymphatics and areolar tissue. As each packet of nodes—paratracheal, pre-esophageal, and subcarinal—is mobilized it should be tacked to a convenient point on the visceral pleura to keep them from obscuring the later dissection. So far the nodes placed in front of the

right innominate vein and superior vena cava have not been included in our dissection, but probably should be.

3. *Inferior pulmonary ligament dissection* (Fig. 6): The vessel at the lower end of the inferior pulmonary ligament is divided, and the esophageal musculature identified above the diaphragm and cleaned of areolar and lymphatic tissue. This packet is elevated upward with the lung to, and then around the inferior pulmonary vein.

4. *Subcarinal node dissection* (Fig. 6): The lung is held anteriorly by steady, gentle traction, elevating bronchi and esophagus, and released momentarily at least every fifteen minutes. Dissection of areolar tissue off the esophagus, sparing if possible the right vagus nerve but dividing its bronchial branches, is continued upward to the level of the carina. The esophagus may then be retracted sharply posteriorly exposing the large conglomerate mass of

Fig. 7.—Right side: Subcarinal lymph nodes dissected onto lung from both bronchi and pericardium. Individual ligation of the bronchovascular elements can now be performed.

lymphoid tissue wedged between the main bronchi. Ready and practically avascular mobilization of this nodal packet is effected by entering the plane adjacent to the posterior points of the left, or contralateral bronchial cartilages. The cartilaginous surface of the bronchus is a safe base for dissection, particularly as the nodes seldom adhere to or overlie the membranous portion of either main bronchus. Dissection is carried to the carina (Fig. 7) and sufficiently along the right main bronchus to allow its transection at the desired level. This still leaves the main mass attached by its lymphatics to the distal bronchus and lung; it remains to lift this packet off underlying pericardium. Here the avascular cleavage plane is immediately adjacent to pericardium.

5. *Anterior pleuro-pericardial dissection:* All lymphatic tissue adjacent to superior vena cava and pericardium around the phrenic nerve is swept laterally, cleanly off these surfaces, and particularly between the pulmonary artery and the

superior pulmonary veins. Unless these nodes which are part of the anterior mediastinal chain are markedly enlarged by inflammatory or metastatic changes or are adherent to pericardium, intrapericardial division of the pulmonary vessels, as practiced by Allison,[1] is not done.

The node dissection, with mediastinal nodes remaining attached to the lung, is now completed, and individual division of the hilar structures can be begun (Overholt[9]).

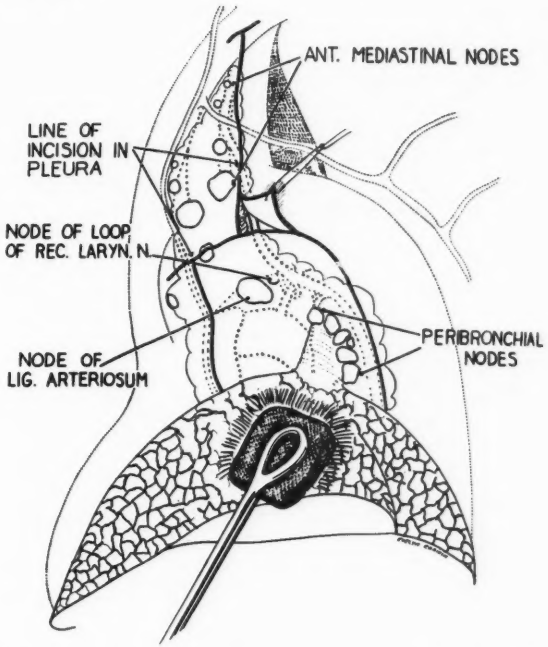


Fig. 8.—Left side: Anatomy of anterior mediastinal and subaortic lymph nodes. The pleural incision is outlined.

Alternatively, the bronchus may be divided before undertaking this subcarinal dissection (Fig. 11) or even completing the dislocation of the node of the arch of the azygos vein or clearing the superior surface of the pulmonary artery. Such an alternative is particularly applicable when a large upper lobe tumor makes access to the region of the pulmonary artery difficult, or when the nodes between the bronchus and pulmonary artery are greatly enlarged. The bronchial arteries are divided prior to bronchial transection. Traction on the distal, severed stump must be gentle to avoid tearing the adjacent nodes prior to their mobilization from the opposite bronchus, pericardium, and pulmonary artery.

Left side.—

1. *Incision* (Fig. 8): This is made into the mediastinal pleura beginning at the apex of the chest and proceeding along the vagus nerve to the arch of

the aorta. Its posterior limb follows the aortic arch to the level of the bronchus and thence medial to that vessel as far as the diaphragm. Its anterior limb extends 1 to 2 cm. anterior to, and inferiorly along the course of the phrenic nerve to the inferior pulmonary ligament to join the posterior incision.

Flaps are elevated and retracted (Fig. 9). The phrenic nerve is fully liberated throughout most of its mediastinal course so that it may be retracted anteriorly and posteriorly by a Penrose drain as the dissection warrants.

2. *Anterior mediastinal dissection:* The apex of the dissection is at the undersurface of the left innominate vein at a point where it crosses the left subclavian artery. The areolar, node-bearing tissue is dissected off the vein, and several small branches must be separately identified and divided. The dissection (Fig. 10) proceeds anteriorly beneath the innominate vein as far as its entrance into the superior vena cava, dividing the highest intercostal and the thymic branches along the way. This is the anterior limit of the dissection.

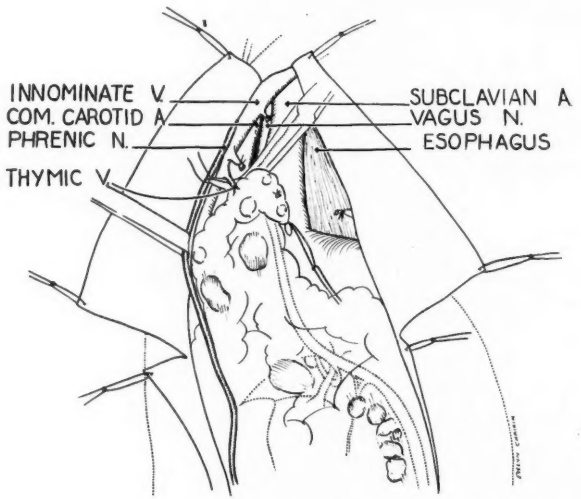


Fig. 9.—Left side: Mediastinal lymph node dissection begun along left innominate vein. Phrenic nerve is retracted anteriorly.

The posterior limit of the dissection is the subclavian artery. The packet of tissue is swept down off the subclavian, common carotid, and innominate arteries and the aortic arch. The vagus nerve is carefully cleaned and, in particular, the large node present in the interval between the common carotid and subclavian arteries is included in the specimen.

3. *Subaortic dissection* (Fig. 11): Beneath the aortic arch, the node of the ligamentum arteriosum and that of the loop of the left recurrent nerve are freed. Lying between the superior border of the pulmonary artery and the anterior surface of the left main bronchus and extending beneath the aorta is a

packet of nodes which must be dissected down toward the specimen. All of these are included with the previously dissected mediastinal packet of tissue and sewn onto the lung surface.

The pleuro-pericardial, inferior pulmonary ligament, and subcarinal dissections (Fig. 12) are similar on both sides and do not warrant special descrip-

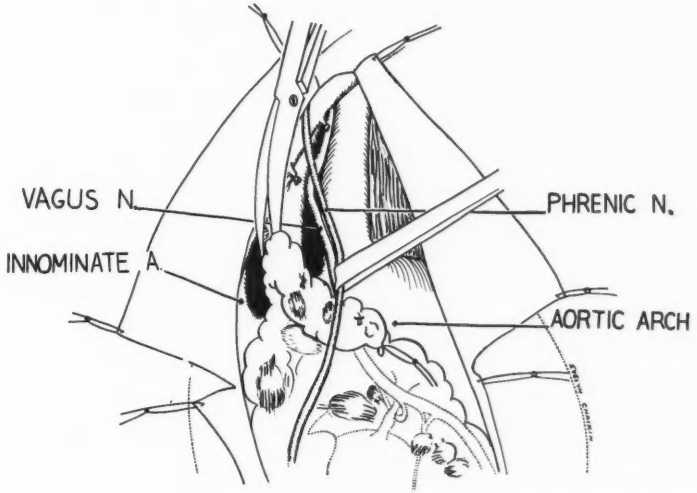


Fig. 10.—Left side: Mediastinal dissection continued. Phrenic nerve retracted posteriorly and lymph node packet passed beneath it.

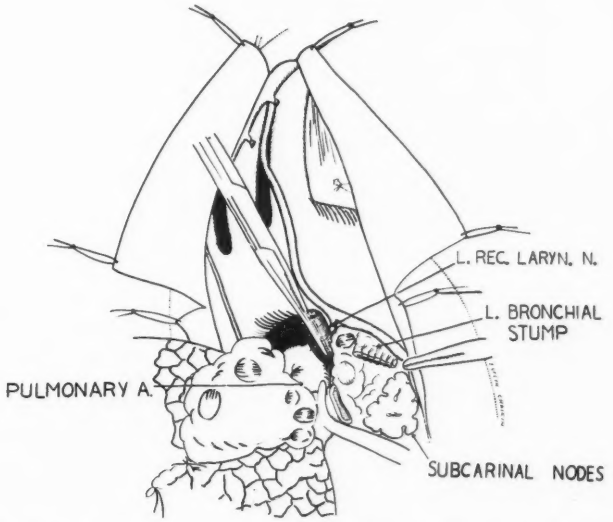


Fig. 11.—Left side: Subaortic lymph node dissection begun *after* bronchus has been divided (alternate method).

tion. The key to dissection in this area is exposure achieved by gentle traction on the lung and mobilization of the esophagus.

Closure of the superior mediastinal pleura at the conclusion of the procedure may aid in reducing subcutaneous emphysema.

MEMORIAL HOSPITAL CASES

Between January, 1949, and April, 1951, sixty pneumonectomies for cancer were performed. Twenty-one of these were simple, and thirty-nine were radical pneumonectomies (Figs. 13, 14, and 15). In the former, one postoperative death due to bronchopneumonia occurred, and in the series of radical pneumonectomies deaths were due to hemorrhage from the pulmonary artery twenty-

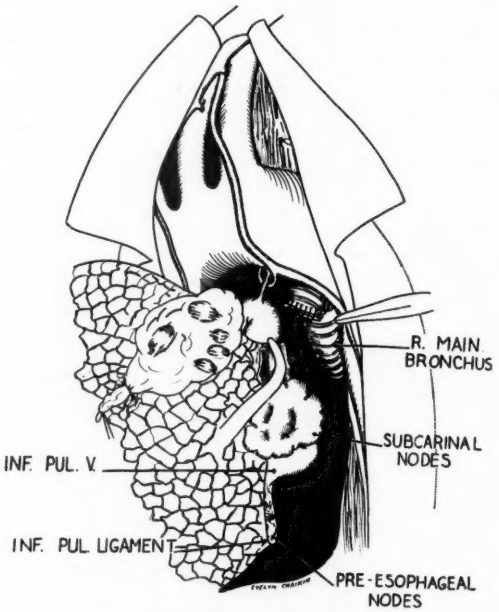


Fig. 12.—Left side: Pre-esophageal and subcarinal lymph node dissection completed. Individual ligation of vascular elements can now be performed.

four hours after operation, massive hemorrhage from a site in the gastrointestinal tract, undisclosed even at autopsy and hemiplegia on the fourth postoperative day. None was charged directly to the more extensive surgery.

In addition to the radical node dissections portions of the pericardium were resected in five instances, and of the chest wall twice. Pericardial resection accompanied simple pneumonectomy only once. The mediastinal dissection does add thirty to forty-five minutes to the operative time, but it is not difficult and, as a matter of fact, greatly aids mobilization of hilar vessels and the bronchus. About two-thirds of our cases have been actually done by resident surgeons.

The only increase in postoperative morbidity is related to the difficulty of maintaining intrapleural pressure at the desired level the first two or three days. Subcutaneous emphysema is prominent, but cardiac arrhythmias have not been more frequent, having been noted seven times following the radical and three after the simple procedure.

In the simple pneumonectomy specimens, intrapulmonic nodes contained metastases ten times, while in the radical pneumonectomy specimens metastases were found twenty-six times in these nodes and eighteen times in the mediastinal group, some of these in the apex of the dissection (Fig. 16). In

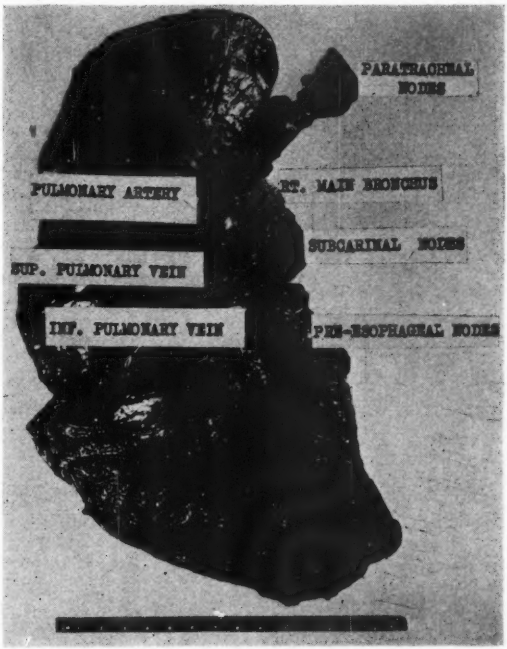


Fig. 13.—Specimen of right radical pneumonectomy, anterior view. Note block dissection.

no specimen were metastases found in the mediastinal nodes without also occurring in the intrapulmonic nodes. In a good many specimens the operator's impression of nodal pathology was not confirmed microscopically.

No safe or true statistical conclusions can be drawn from so few cases and in the absence of a long follow-up period. For instance, the fact that as of April 1, 1951, nine of the twenty-one patients with simple pneumonectomies are already dead of their cancer, whereas only eleven of the thirty-nine radically operated patients have died, cannot serve as evidence in favor of radical pneumonectomy.

RATIONALE

When a primary carcinoma of the lung is centrally placed, either arising in or extending to a main-stem bronchus, the minimal procedure for total removal is a pneumonectomy. The same general rule holds true for a more peripherally placed carcinoma of the lung that invades other lobes by direct extension or by intense inflammatory reaction. Although the necessity for a complete lung removal, when feasible, is not questioned in this group, clarification is needed as to the advisability and the extent of an accompanying lymph node dissection.

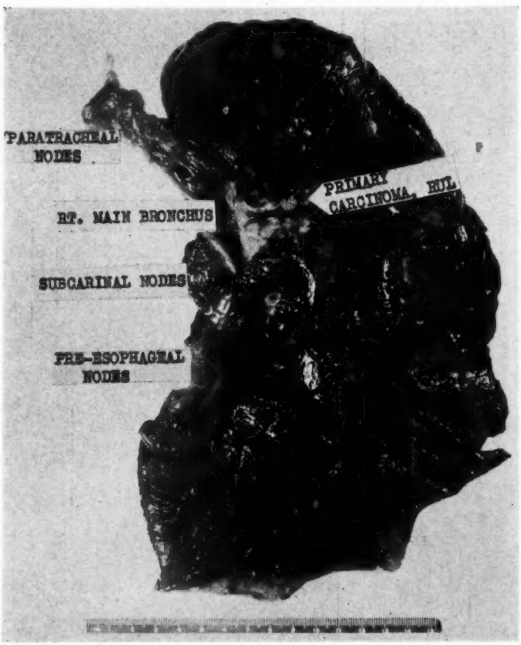


Fig. 14.—Specimen of right radical pneumonectomy, posterior view. Note block dissection. and primary carcinoma.

When a primary carinoma of the lung is confined to a lobe, is peripherally placed, and is relatively small and well circumscribed, the surgeon may elect to do a less radical procedure on the grounds that he is sparing useful respiratory surface while still performing an adequate cancer operation. However, Most, as quoted by Rouviere, has said that "pulmonary lobes do not establish distinct lymphatic regions. The lymphatic tracts which run between the lobes are fed by lymph vessels from the two neighboring lobes." This seems certainly suggested by the size of the interlobar venous channels which may well be accompanied by lymphatics, and also the frequent failure of complete division of lobes anatomically. It is possible that the intrapulmonary lymph

nodes which drain a particular lobe may be situated within the substance of another and, although the original lobe containing the primary focus of cancer is removed, its metastatic deposit could be left behind. If a lobectomy is elected it should be accompanied by a node dissection as radical as possible which takes into consideration those that may be involved knowing the location of the primary tumor. The dissection should include the subcarinal and the paratracheal groups, and also remove nodes about adjacent bronchi.

Small or peripheral cancers have been found to metastasize to hilar and mediastinal nodes. It is, therefore, encumbent upon us to be adequate and deal radically with these small lesions in the hope that at this one procedure, at which time the chance to cure the patient is optimum, all possible cancer and its potential areas of spread are removed.

Fig. 15.—Specimen of left radical pneumonectomy. The pre-esophageal group has not been labeled.

Although lesions of the lingula and left lower lobe are less amenable to a complete lymph node dissection for reasons already given, the excision of subcarinal nodes and those of the ipsilateral paratracheal group offers a better chance of including random metastases than if it were not done.

When a lesion is in doubt as to its being primary or metastatic, we do an aspiration biopsy through the open thorax[2] or resect a lobe and have a frozen section biopsy performed. If it then proves to have its origin in the lung, a radical pneumonectomy is completed at that time.

Koletsky[7] has noted that all of the various histologic types of carcinoma of the lung, whether centrally or peripherally located, are capable of metastasizing to the regional lymph nodes. The squamous carcinomas arising in the main bronchi seem actually to remain within the thorax, and in 65 per cent of the cases killed by local invasion and extension rather than by distant metastases. The terminal bronchiolar and adenocarcinomas comprise most of the peripheral lesions. Along with the anaplastic or oat-cell carcinomas, these are infamous for the rapidity of their vascular, extrathoracic spread, but they, too, metastasize to the regional lymph nodes with some regularity. Although the over-all prognosis for these more rapidly disseminating histologic

Fig. 16.—Specimen of right radical pneumonectomy showing primary carcinoma of the right lower lobe with metastases to the subcarinal ("hilar") and paratracheal ("superior mediastinal tracheal") lymph nodes.

types is poor, nevertheless when the indications for resectability exist, the radical operation should be carried out. For it is felt that the histologic type of the tumor should not be the influencing factor on the procedure to be performed, as five-year survivals in all types have been reported.

In various communications it has been stated that the reason for leaving the hilar and mediastinal lymph nodes intact is that they felt clear of metastases.[3] Or else, one so-called representative lymph node was removed for frozen section and, when reported as negative, the others were presumed to

be negative as well. Some feel that the finding of one positive lymph node warrants leaving the rest in situ, and then they do a "palliative resection." The term "palliative" used in this sense has an inherent defeatism which is at variance with the fact that there have been five-year survivals of primary lung cancer with proved metastatic hilar lymph nodes. Graham's[6] first case has gone eighteen years after the pneumonectomy specimen showed lymph nodes positive for metastases. The term also presupposes that there is certainty of metastatic lymph node involvement rather than enlargement of these structures because of other conditions.

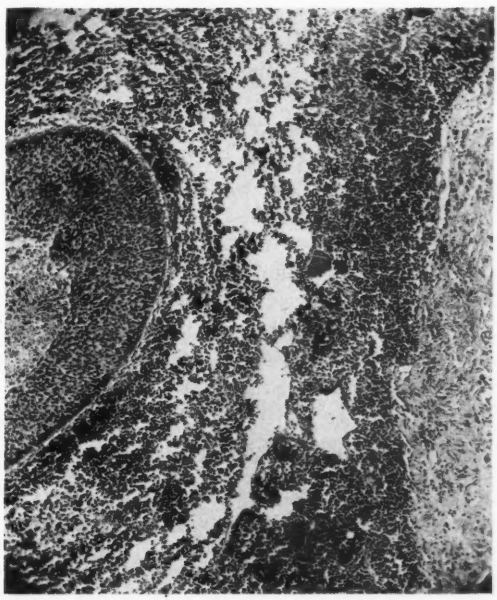


Fig. 17.—Small focus of metastatic epidermoid carcinoma of the lung in one paratracheal lymph node out of eight that were sectioned.

It is believed by the authors that it is impossible to determine the presence of metastases in a lymph node by gross observation or palpation. In fact, even the "microscope can err" in this regard. For example, Saphir and Amromin[11] re-examined axillary lymph nodes from thirty radical mastectomy specimens that had been reported uninvolved on routine examination. They found metastasis in one-third on serial sectioning of the nodes. In our own experience metastatic foci have been observed in part of one high-powered field of a lymph node section (Fig. 17). Certainly no surgeon can claim a microscopic touch of such delicacy!

If judgment as to whether or not a radical lymph node dissection is to be performed is based upon the node taken for frozen section, it must be remembered that: (a) the correct node or nodes must always be selected; (b)

the microtome knife must pass through the metastatic focus; and (e) the occasional skipping of nodes in a chain makes all of them suspect and not just one alone.

The excision of all possible lymphatic areas by radical node dissection largely removes the element of conjecture about their contents. It is our belief that if there are no marked medical contraindications, a radical pneumonectomy be performed for all primary carcinomas of the lung. All procedures short of this may make a compromise with cure.

SUMMARY

1. A lung can be removed in a block dissection with most of its hilar and mediastinal lymph nodes. This procedure is called a radical pneumonectomy, and is differentiated from a simple pneumonectomy, the removal of the lung alone.

2. The procedure is based upon available knowledge of the anatomy and direction of flow of the lymphatic system of the lung.

3. From a pathologic view, this radical dissection is considered to be sound therapy for primary cancer of the lung, as the regional lymph nodes are involved frequently with metastases from both central and peripheral tumors of all histologic types; this involvement can only be determined accurately and with certainty by microscopic observation.

4. In two and one-half years, thirty-nine radical pneumonectomies have been done. Lymph nodes positive for metastases beyond the limits of the average simple pneumonectomy were found in eighteen cases.

5. It is felt that experience will clarify whether such a radical node dissection is a factor in survival for carcinoma of the lung.

REFERENCES

1. Allison, P. R.: Discussion Following the Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20:** 349-365, 1950.
2. Cahan, W. G., Butler, F. S., Watson, W. L., and Pool, J. L.: Multiple Cancers; Primary in the Lung and Other Sites, J. THORACIC SURG. **20:** 335-348, 1950.
3. Churchill, E. D., Sweet, R. H., Soutter, L., and Scannell, J. G.: The Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. **20:** 349-365, 1950.
4. Crafoord, C.: On the Technique of Pneumonectomy in Man, Acta Chirurgica Scandinavica **81,** Suppl. LIV, 1938.
5. Drinker, C. K.: Lane Medical Lectures, The Lymphatic System, Stanford University, Calif., 1942, Stanford University Press, pp. 73-74.
6. Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of the Bronchus, J. A. M. A. **101:** 1371, 1933.
7. Koletsky, S.: Primary Carcinoma of the Lung—A Clinical and Pathology Study of 100 Cases, Arch. Int. Med. **62:** 636-651, 1938.
8. Ochsner, A.: Significance of Metastasis in Primary Carcinoma of the Lung, J. THORACIC SURG. **11:** 357-387, 1942.
9. Overholt, R. H., and Langer, L.: The Technique of Pulmonary Resection, Springfield, Ill., 1949, Charles C Thomas, Publisher.
10. Rouviere, H.: Translated by M. J. Tobias, Anatomy of the Human Lymphatic System, Ann. Arbor, Mich., 1938, Edwards Bros., Inc.
11. Saphir, O., and Amromin, G.: Obscure Axillary Lymph Node Metastases in Carcinoma of the Breast, Cancer **1:** 238-241, 1948.
12. Sweet, R. H.: Thoracic Surgery, Philadelphia, 1950, W. B. Saunders Company.
13. Weinberg, J., and Greaney, E. M.: Identification of Regional Nodes by Means of a Vital Staining Dye During Surgery of Gastric Cancer, Surg., Gynec. & Obst. **90:** 561-568, 1950.

ADDENDUM

The operation as above described has been modified in several ways since its presentation.

On the right side, the procedure of excision of the right paratracheal nodes has been facilitated and expedited by sectioning, and including in this lymph node packet the vagus nerve inferior to the origin of its recurrent laryngeal branch. This requires careful identification of this branch and has not, in at least ten instances, produced any noticeable alteration in postoperative cardiac or gastrointestinal function.

On the left side, two alterations are under consideration:

1. The ductus arteriosus is ligated and divided to permit more thorough dissection in the area just beneath the arch of the aorta.
2. The first two pairs of intercostal arteries arising from the aorta are ligated and divided, permitting mobilization of the arch to allow dissection of the left paratracheal nodes that run between the arch and the trachea. This is a suggestion of Professor Pietro Valdoni of Rome, Italy.

DISCUSSION

DR. WILLIAM L. WATSON, New York.—Perhaps some of the points made by Dr. Cahan could be emphasized. He pointed out that no claim is made for originality as to the concept of an en bloc removal of a cancerous lung and its accessible lymph nodes, but he has attempted to focus attention on the fact that the operation is not only a feasible one, but is attended by an acceptable operative mortality rate of 7.7 per cent. It is to be noted also that in this series of cases two-thirds of the operations were carried out by our resident surgeons, well trained in general surgery, but some of whom have had less than three months of special training in thoracic surgery. Perhaps it should be restated that the operation of radical pneumonectomy as described by Dr. Cahan is reserved for the fair operative risk patients, and it is significant that during the period under discussion the service performed twelve lobectomies for cancer in patients who were medically unsuitable for the more radical procedure.

Not included in the statistics presented by Dr. Cahan is one case of mistaken diagnosis. A radical pneumonectomy with lymph node dissection was carried out for what proved to be an atypical form of pulmonary tuberculosis. The point of interest is that the excised lymph nodes contained active and caseating tuberculosis, leading to a question of what the future of those lymph nodes might be in relation to the possibility of contralateral postoperative spread. It might be said that in this case there was no postoperative spread. As Dr. Cahan has stated, we do not know what the ultimate salvage rate will be, but it is my hope that the radical pneumonectomy will add somewhat to the expected survival rate, but time will be required for final evaluation.

DR. J. F. HIGGINSON, Portland, Ore.—We have found this paper of Dr. Cahan's very interesting. A truly ideal cancer operation is not possible for cancer of the lung, as is the case in the breast or the neck or elsewhere. Of necessity, compromises must be made, essential structures left undisturbed, and the dissection often carried very close to the actual primary malignancy or its nodal metastases or local spread. Realizing these facts, it seemed to us that the best possible block dissection, with the least compromise, would be the most nearly ideal cancer operation in dealing with bronchiogenic carcinoma. We began planning and doing in our private services and the thoracic surgical service at the Portland, Oregon, Veterans Hospital, a block node dissection in July of 1948, and since that time we have had occasion to do forty-two pneumonectomies for bronchiogenic carcinoma. Of these, thirty-eight have had partial or complete node-bearing tissue block dissections as part of the pneumonectomy. Of the thirty-eight, ten were partial and twenty-eight complete. By partial is meant that the superior mediastinal node-bearing tissue along the trachea was left undisturbed. In all thirty-eight cases the nodes of the inferior posterior mediastinum, the homolateral hilar nodes, the subbifurcation nodes, and as many as possible of the contralateral hilar nodes were removed as part and parcel of the specimen. In most of these cases, various portions of diaphragm, mediastinal pleura

and pericardium, and segments of phrenic and vagus nerves and chest wall were also removed, as is common. In some instances, portions of esophageal musculature and cuffs of the left atrial wall were removed.

We have not as yet attempted to evaluate these cacses from the standpoint of early or late results, but the pathologic findings are interesting. Of the thirty-eight cases, sixteen had nodes and twenty-two had positive nodes. If we consider only the twenty-eight complete block dissections, eleven had negative nodes and seventeen had positive nodes.

(Slide.) This is a section of a man, right side, demonstrating, as you saw in the slides of the essayists, the paratracheal superior mediastinal lymph node block; the other nodes as a complete block turned up from the contralateral bronchus, sub-bifurcation area, and homolateral bronchus are included.

(Slide.) This is another right-sided lesion without a card to make the nodes stand out; the nodes are tossed up over the top of the specimen. You can see the other nodes gathered together in a smooth continuous clump next to the bronchus.

(Slide.) Another right-sided lesion, again demonstrating that this group of node-bearing tissue can be removed en bloc with the lung.

(Slide.) This is a left-sided lesion demonstrating pericardium and the inferior pulmonary vein; this vein was totally occcluded by carcinoma and this was bulging into the left atrial lumen as a polypoid thumblike mass. The nodes in this man's specimen were negative.

DR. JOHN M. CHAMBERLAIN, New York.—I did not intend to discuss this paper, but I find it extremely interesting and worth while, and so I rise mainly to raise one question and to invite discussion from the authors.

Since Allison and Bloch first called this type of resection to our attention, we have been trying a more radical procedure, but have wondered whether we should leave the lung in and do the radical dissection first, or whether we should remove the lung and do the radical mediastinal dissection later. If the mediastinal dissection is done first, extensive manipulation of the lung is necessary. Should the vein be invaded, this varied dislocation of the lung could conceivably dislodge small emboli into the systemic circulation. All of us, I am sure, occasionally have found the veins invaded at operation and have worried, therefore, about the dissemination of such emboli. Perhaps veins within the parenchyma are always invaded. On the other hand, Drinker has shown that ligation of the veins tremendously increases the peribronchial lymph flow. We have therefore determined operability with as little manipulation of the lung as possible, and then proceeded to clamp and divide the bronchus immediately, ligating the vessels next. After removal of the specimen, the radical dissection is accomplished.

Today, Dr. Cahan pointed out that they begin their radical dissection in the mediastinum. So I raise this point: I wonder which is the more hazardous as a result of the operation—dissemination via the veins through excessive manipulation of the lungs, or via the lymphatics, the most distant of which are interrupted first, as outlined by the authors.

DR. ADRIAN LAMBERT, JR., New York.—I, too, should like to compliment Dr. Cahan on his fine presentation. I wish that the actual procedure were as easy as some of these pictures portray. In our experience it is much more difficult to dissect out the mediastinum than these drawings would have us believe.

This procedure is one that should be performed in cases in which the patient can stand the operation. At Bellevue we have employed radical mediastinal excision when this is the case, and have been doing it more and more in routine removal of carcinoma of the lung. There are one or two points I should like to make. First, the value of this procedure will depend on the relation of five-year survival to the type of cases selected for radical dissection. In cases in which we cannot remove the entire lung for functional reasons, where the patient cannot stand the entire dissection—and there are cases we encounter in which that is so—it is wise to bear in mind one or two points which may be of value. One is that carcinoma of the upper lobe may metastasize to the carinal node, so that

if the mediastinal extension is to be removed, that node should be removed too. Carcinoma of the lower lobe more frequently goes to the lower nodes, so that in doing a partial resection that part should be removed first. Carcinoma of the upper lobe, in my experience, does not go to the lower nodes down toward the diaphragm without first extending upward, so that in doing a partial dissection, this is worth bearing in mind.

The third point I should like to mention is the presence of a structure which is not emphasized in the anatomy books. From studies of bodies that I have dissected and patients encountered at the operating table, there seems to be a fibrous membrane situated longitudinally posterior to the trachea extending down to the esophagus. This seems to be a constant finding and appears to prevent extension in the mediastinum, in this area at least, from one side to the other.

One note about complications: These patients are prone to develop mediastinal emphysema which should be controlled with negative intrathoracic pressure or suction. In addition to having difficulty coughing, and a large amount of secretions, they also seem to develop edema of the opposite lung, which brings up the question of whether the lymph drainage to the opposite lung may be impaired. I do not feel that it has been determined how compatible with life it is if you remove all the peribronchial nodes on the other side.

I feel that this procedure should be done more frequently, and I want to compliment the men from Memorial on directing attention to its more routine use.

DR. CAHAN (closing).—I would like to thank Dr. Watson, Dr. Higginson, Dr. Chamberlain, and Dr. Lambert for their discussions, and to take up briefly some of the points they have raised.

Dr. Higginson mentioned the point that Dr. Churchill quoted last year about there being a morass of lymphatics in the mediastinal area. To be sure that is so, but we like to emphasize that these can be removed for the most part en bloc, and although we do probably make certain compromises with certain lymphatics, I feel that this is certainly true also in operations performed on the breast in which there are parasternal lymph nodes as possible foci of metastases; also in the stomach and colon metastatic spread may well extend beyond the standard limits of resection.

Dr. Chamberlain's question of sequence of operation—doing the pulmonary vein or the bronchus first—is something about which we could talk at great length. We apply the principle of cancer surgery and take the distalmost point of possible lymphatic spread first, working back toward the primary itself. The question of avoiding venous emboli is a good point, and I think we should consider it carefully.

With Dr. Lambert's statement about the simplification of our pictures I agree wholeheartedly. But I am sure that the people in the back row, if all the ties and fibrous tissue were inserted into the picture, would be indeed confused. I agree that there are points in the dissection that are more difficult than others, but these can be surmounted with patience. But basically, when you get that lymphatic packet, for example in the paratracheal or retrocaval areas on the right side, it does seem to come down with relative ease and well contained, particularly if you hug close to the major vessels and to the trachea. We have not seen the edema of the opposite lung noted by Dr. Lambert.

PLEUROPULMONARY RESECTION WITH HEMIDIAPHRAGMECTOMY

Preliminary Report

Bert H. Cotton, M.D., and Joao R. F. Penido, M.D. (by invitation)
Beverly Hills, Calif.

THE present paper consists of a discussion of indications, complications, technical problems, and results in a series of twenty-eight cases of coexisting pulmonary and pleural disease treated by surgical methods. Each of the twenty-eight patients received pulmonary resection combined with pleurectomy. In eleven instances, subtotal or total hemidiaphragmectomy was performed as an adjunct to pleuropulmonary resection. The results obtained are encouraging.

PLEUROPULMONARY RESECTION

For many years chest surgeons have performed pneumonectomy with pleurectomy merely for reasons of technical convenience. Pleuropulmonary resection has been employed successfully in the management of tuberculosis with complicating empyema. However, little use has been made of this combined procedure in the treatment of malignancy. Our experience indicates that pleuropulmonary resection may prove lifesaving in cases of bronchial carcinoma heretofore considered hopeless and will increase the present salvage rate in routine resectable cases.

HEMIDIAPHRAGMECTOMY

The value of subtotal or total hemidiaphragmectomy as an adjunct to the treatment of thoracic malignancy also has not been fully appreciated. If the malignant process involves the diaphragmatic pleura, either by direct extension or by implants, we have found a hemidiaphragmectomy combined with pleuropneumonectomy makes the operation more definitive for removal of all neoplastic implantations. In our opinion, previous reports in the literature have placed too great importance on preserving the continuity of the diaphragm. Our experience is that a subtotal or total hemidiaphragmectomy can be performed without detrimental effects on the patient.

The fate of the diaphragm depends upon the stage of pleural involvement. If no involvement has occurred because of a localized pocket of pleural fluid, the diaphragm is allowed to remain intact. Pleuropulmonary resection with subtotal hemidiaphragmectomy is performed when segmental involvement of the diaphragm is present. If the entire diaphragm is involved, complete hemidiaphragmectomy is the treatment of choice.

In a large percentage of our patients we have found it impossible to remove the pleura from the diaphragm without causing rupture into the pleural space. The dissection thus is facilitated by removal of the diaphragm.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

One complication of hemidiaphragmectomy is the pronounced shortness of breath. We have surmised that this manifestation is due to loss of the central attachment of the contralateral leaf of the diaphragm. The shortness of breath remains for about seven to ten days postoperatively, after which the mediastinum is stabilized. Apparently the thick fibrinous membrane which is formed over the peritoneum acts as a buffer as well as a solid attachment for the remaining leaf of the diaphragm.

In cases of tuberculosis with empyema, the danger of abdominal infection following pleurodiaphragmectomy may be avoided by preserving the peritoneum; this structure provides a base upon which a fibrinous membrane rapidly forms. Recurrent empyema or bronchopleural fistula will be late postoperative complications, allowing an interval during which a heavy fibrinous wall forms between the abdominal and thoracic cavities.

ANALYSIS OF AUTHORS' CASES

Tables I, II, and III show the types of cases included in the present study and the treatment employed in each type. The twenty-eight instances may be divided into two major classifications, that of malignancy and that of infection. Surgical treatment in cases of the infective type is less radical than in malignancy, where only wide excision can improve the prognosis.

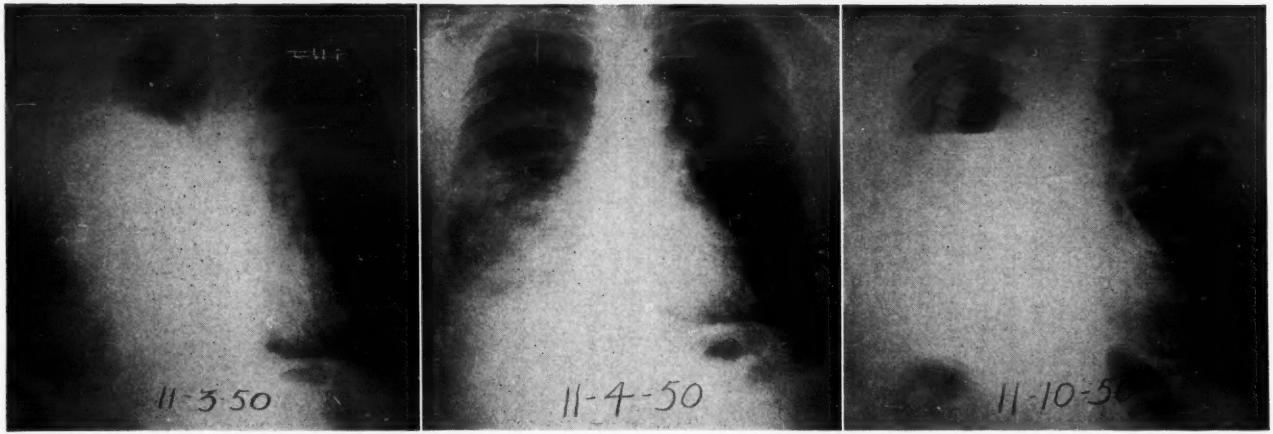
TABLE I

1. Primary carcinoma of the lung with associated pleural effusion and malignant cells	8
2. Diffuse primary neoplasm of the pleura	2
3. Pulmonary tuberculosis with tuberculous empyema	9
4. Pulmonary fungus cavities with rupture and empyema	8
5. Bronchiectasis with empyema	1

1. *Primary Carcinoma of the Lung With Associated Pleural Effusion and Malignant Cells* (8 cases).—When malignancy of the lung has involved the parietal or visceral pleura by direct extension or implantations with or without pleural effusion, pleuropulmonary resection with pleurodiaphragmectomy often will decrease morbidity and mortality. In such instances this combination of operations may be considered a routine procedure unless a protective cushion of lung is present between the lesion and the pleura.

Five of the eight patients are alive and well after postoperative periods of two years, one year, nine months, six months, and four months, respectively. Two patients died with metastasis less than a year after operation. One 72-year-old white woman died of coronary occlusion twenty-four hours postoperatively.

2. *Diffuse Primary Neoplasm of the Pleura* (2 cases).—This neoplasm, known as diffuse mesothelioma or endothelioma of the pleura, involves the entire pleura and usually ensheathes the lung in tumor tissue of the diffuse type. The two such cases in the present series were treated by pneumonectomy with hemidiaphragmectomy. One patient presented erosion through the

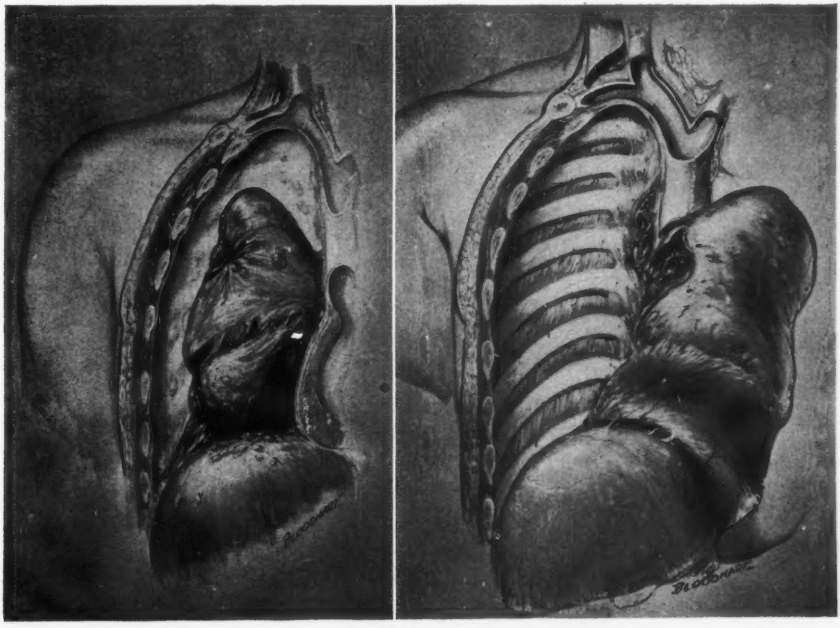


A *B.* *C.*

Fig. 1.—(Case 6) *A*, Primary carcinoma of the right lung with associated pleural effusion and malignant cells; *B*, removal of fluid with demonstration of the small lesion in the midlung area; *C*, second postoperative day, no abdominal viscera in right hemithorax.

diaphragm with involvement of the left adrenal gland and left kidney; these organs also were resected. The patient progressed well for fourteen months postoperatively, then died of cerebral vessel rupture. The second patient lived seven months after operation and died of abdominal metastasis.

3. *Pulmonary Tuberculosis With Empyema* (9 cases).—In our experience surgical treatment of pulmonary tuberculosis with positive sputum in combination with tuberculous empyema has been discouraging. Our disappointment may be attributed to: (1) a high mortality rate; (2) the *long, complicated surgical regimen* necessitating skeletal deformity; and (3) failure to control pulmonary disease due to the extrapulmonic heavy fibroblastic membrane over the pleura.



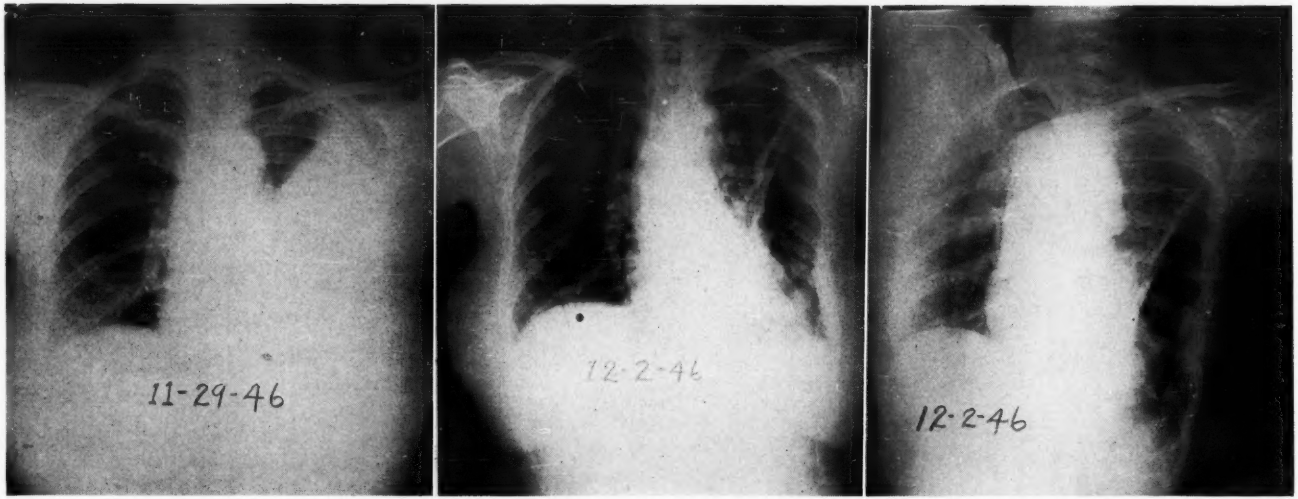
A. *B.*

Fig. 2.—(Case 6) *A*, The appearance of the lesion at the operating table; *B*, pleuropneumonectomy with hemidiaphragmectomy.

Thoracoplasty treatment is directed primarily toward cure of the empyema. It is hoped that the pulmonary disease also is cured. Extrapleural pulmonary resection is definitive for both pulmonary and pleural tuberculosis.

All of the nine patients recovered. One patient developed a late bronchopleural fistula, treated successfully with sodium hydroxide cautery, drainage, and thoracoplasty.

4. *Fungus Disease of the Lung With Ruptured Cavity and Empyema* (8 cases).—Pleuropulmonary resection is proving of value in the management of fungus diseases of the lung and pleura. In our series of sixty cases of pulmonary fungus disease treated by surgical means, eight patients exhibited



A. *B.* *C.*

Fig. 3.—(Case 10) *A*, Diffuse primary neoplasm of the left pleura with pleural effusion and malignant cells; *B*, removal of the fluid with diagnostic pneumothorax, neoplasm demonstrated; *C*, oblique view showing the multiple tumor growths of the pleura.

coexisting pleural and pulmonary involvement. Each of these eight patients received pleuropulmonary resection, recovered with no complications, and returned to normal life.

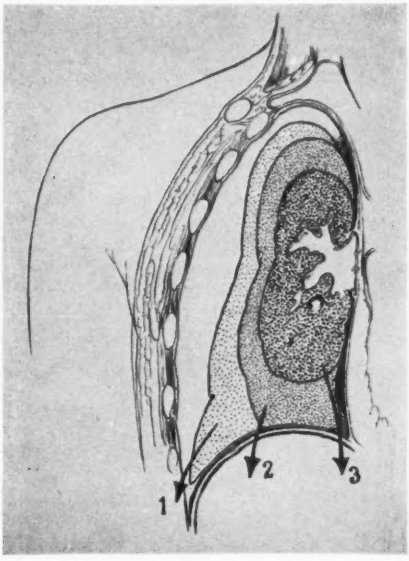


Fig. 4.—The fate of the diaphragm depends upon the pleurodiaphragmatic involvement. 1. Hemidiaphragm remains intact. 2. Subtotal hemidiaphragmectomy. 3. Hemidiaphragmectomy.

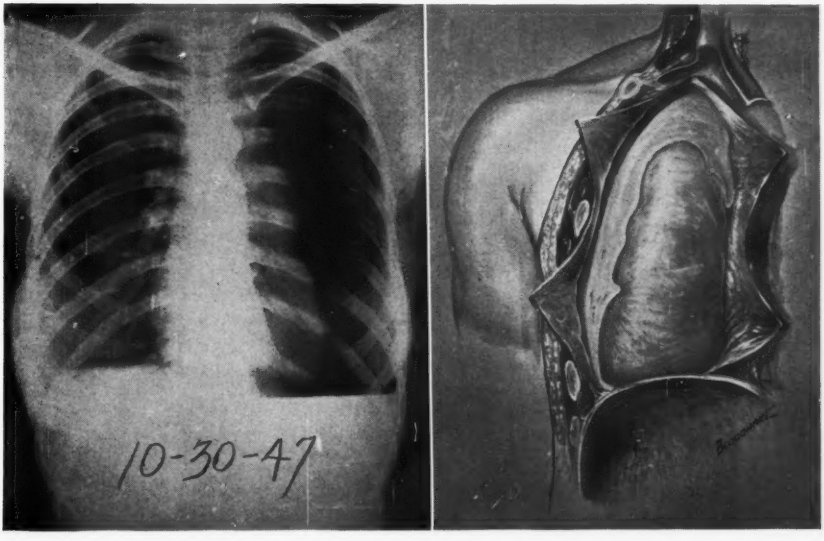
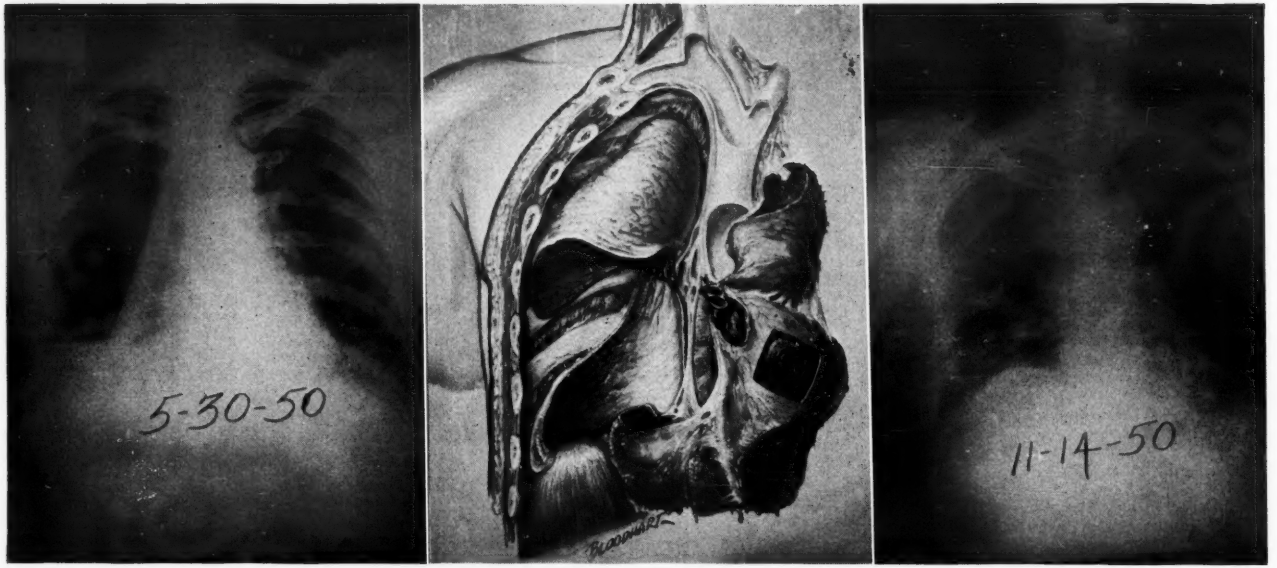


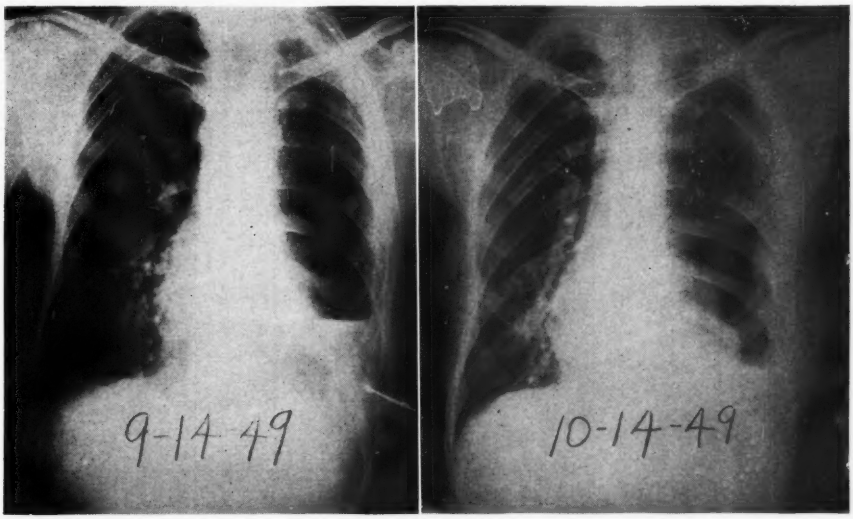
Fig. 5.—(Case 18) *A*, Active pulmonary tuberculosis of the left lung with tuberculous empyema; *B*, appearance of the diseased lung and pleura at the operating table. Pleuropneumonectomy was performed.



A. *B.* *C.*

Fig. 6.—(Case 19) *A*, Pulmonary tuberculosis and coccidioidomycosis of the right middle lobe, tuberculous and coccidioidal empyema was also present; *B*, right pleurolobectomy of the middle lobe which also included decortication of the right upper and lower lobes; *C*, postoperative films with right upper and lower lobes reexpanded.

5. *Bronchiectasis With Bronchopleural Fistula and Empyema* (1 case).—Profound disagreeable symptoms, periodic incapacitation, and early death has been the fate of patients with bronchiectasis complicated by bronchopleural fistula and empyema. The one such patient in our series developed a lung abscess which ruptured, resulting in suppurative empyema with bronchopleural fistula. Closed catheter drainage was present when the patient was first seen by us. Left pleuropneumonectomy was performed after which the patient progressed satisfactorily until a bronchopleural fistula developed on the fourteenth postoperative day. This was treated successfully by drainage, sodium hydroxide, cauterization, and thoracoplasty. The patient has now returned to work.



A. *B.*

Fig. 7.—(Case 28) *A*, Bronchiectasis of the left lung with bronchopleural fistula and empyema; *B*, postoperative film one week following left pleuropneumonectomy.

TABLE II. SURGICAL PROCEDURES EMPLOYED

1. Pleuropneumonectomy	5
2. Pleuropneumonectomy and hemidiaphragmectomy	5
3. Pleuropneumonectomy and segmental diaphragmectomy	6
4. Pleurolobectomy	6
5. Segmental pleurolobectomy	6
Total	28

SURGICAL TECHNIQUE

Through the bed of the resected rib, an extrapleural plane of dissection is found and enlarged sufficiently to permit the use of a rib spreader. A small incision is made in the parietal pleura, and the intrapleural contents explored. The fluid content is aspirated; this precaution prevents widespread contam-

TABLE III. PRIMARY CARCINOMA OF THE LUNG WITH ASSOCIATED PLEURAL EFFUSION AND MALIGNANT CELLS

CASE	AGE (YR.)	SEX	RACE	DIAGNOSIS CONFIRMED BY	EXTENT OF INVOLVEMENT	CHARACTER OF FLUID	OPERATIVE PROCEDURE	COMPLICATIONS	PRESENT CONDITION
#1 W. S.	57	M	W	Malignant cells in pleural space	Total pleura involved	Amber	Left pleuropneumonectomy with hemidiaphrectomy and resection of chest wall	P.O. shortness of breath	Living P.O. 2 yr.
#2 M. M.	69	F	W	Malignant cells in pleural space	Total pleura and one-half of left diaphragm involved	Amber	Left pleuropneumonectomy with subtotal hemidiaphragmectomy and resection of chest wall	P.O. shortness of breath	Living P.O. 1 yr.
#3 I. C.	51	M	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Left pleuropneumonectomy with subtotal hemidiaphragmectomy	P.O. shortness of breath	Living P.O. 9 mo.
#4 F. D.	67	W	M	Malignant cells in pleural space	Total pleura, no diaphragm involved	Amber	Right pleuropneumonectomy with chest wall resection	None	Living P.O. 4 mo.
#5 E. O.	53	M	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Right pleuropneumonectomy with subtotal hemidiaphragmectomy	None	Living P.O. 4 mo.
#6 A. B.	66	M	W	Malignant cells in pleural space	Total pleura and all of diaphragm involved	Amber with blood	Right pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 4 mo. extension of disease
#7 J. P.	61	M	W	Malignant cells in pleural space	Total pleura and all hemidiaphragm involved	Amber	Left pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 8 mo. extension of disease

#8 N. L.	72	F	W	Malignant cells in pleural space	Total pleura and one-third of diaphragm involved	Amber	Right pleuropneumonectomy with subtotal hemidiaphragmectomy	Coronary occlusion	Dead P.O. 24 hr.
					Diffuse Primary Neoplasm of the Pleura				
#9 F. R.	41	M	W	Malignant cells in pleural space	Total pleura and all of hemidiaphragm involved	Amber	Left pleuropneumonectomy with hemidiaphrectomy and left nephrectomy and adrenalectomy	P.O. shortness of breath	Dead P.O. 14 mo.
#10 B. C.	32	F	W	Malignant cells in pleural space	Total pleura and all of hemidiaphragm involved	Amber	Right pleuropneumonectomy with hemidiaphragmectomy	P.O. shortness of breath	Dead P.O. 7 mo.
					Pulmonary Tuberculosis With Tuberculous Empyema				
#11 G. H.	26	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura, no diaphragm involvement	Purulent	Right pleuropneumonectomy	None	Alive and well
#12 D. E.	37	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura, no diaphragm involvement	Purulent	Right pleuropneumonectomy	None	Alive and well
#13 R. T.	40	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and one-third of diaphragm involved	Purulent	Right pleuropneumonectomy and subtotal hemidiaphragmectomy	Bronchial fistula developed 2 mo. P.O.	Alive and well
#14 I. A.	28	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm involvement	Amber cloudy	Left upper pleurolobectomy. Decortication of lower lobe	None	Alive and well

TABLE III—CONT'D

CASE	AGE (YR.)	SEX	RACE	DIAGNOSIS CONFIRMED BY	EXTENT OF INVOLVEMENT	CHARACTER OF FLUID	OPERATIVE PROCEDURE	COMPLICATIONS	PRESENT CONDITION
#15 W. E.	53	M	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm involvement	Purulent	Left pleuropneumonectomy	None	Alive and well
#16 R. B.	48	F	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura and one-fourth diaphragm involved	Purulent	Right upper pleurolobectomy. Decortication of lower lobes	None	Alive and well
#17 I. U.	45	F	W	Sputum positive for tuberculosis and coccidioidomycosis. Pleural fluid positive for tuberculosis	Subtotal pleura and no diaphragm involvement	Purulent	Right upper pleurolobectomy. Decortication	None	Alive and well
#18 A. B.	39	F	W	Sputum and pleural fluid positive for tuberculosis	Subtotal pleura involved	Purulent	Right middle pleurolobectomy	Drainage from previous wound for 4 mo.	Alive and well
#19 E. G.	50	F	W	Sputum and pleural fluid positive for tuberculosis and coccidioidomycosis	Subtotal pleura involved	Purulent	Right middle pleurolobectomy	None	Alive and well
Pulmonary Fungus Cavities With Rupture and Empyema									
#20 G. W.	49	M	W	Pleural fluid positive for coccidioidomycosis	Subtotal pleural involvement	Purulent	Right lower pleurolobectomy	None	Alive and well

#21 R. L.	36	F	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right lower pleurolobectomy	None	Alive and well
#22 L. M.	36	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left upper segmental pleurolobectomy	None	Alive and well
#23 D. B.	41	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right upper segmental pleurolobectomy	None	Alive and well
#24 J. T.	19	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left upper segmental pleurolobectomy	None	Alive and well
#25 L. H.	19	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left lower segmental pleurolobectomy	None	Alive and well
#26 C. B.	44	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Right upper segmental pleurolobectomy	None	Alive and well
#27 H. R.	43	M	W	Pleural fluid positive for coccidioidomycosis	Total pleural involvement	Purulent	Left lower segmental pleurolobectomy	None	Alive and well
					Bronchiectasis Wtih Empyema				
#28 H. G.	40	M	W	Mixed infection	Total pleural involvement	Purulent	Left pleuropneumonectomy	Bronchial fistula 10 days P.O.	Alive and well. Fistula healed

ination if the pleura is inadvertently opened during the dissection. If pulmonary resection is decided upon, the pleural wound is closed with a running suture, inverting the edges. The extrapleural plane is then dissected around the entire chest cavity, down to the diaphragm, over the apex, and down the mediastinal surface to the hilar structures. In instances of involvement of the diaphragm, the insertions are dissected away from the chest wall by blunt dissection; it is sometimes easier to start with the lateral insertions. The peritoneum is located and left intact while the diaphragm is dissected away. The medial attachment is divided beyond the pleura in the mediastinum. On the right side, the medial attachment occasionally is freed from the inferior vena cava only with difficulty. In malignant cases, if there is involvement around the hilum, or if the distal portions of the vessels seem to be involved, the pericardium is entered and the vessels secured inside the pericardium. If the pleura cannot be separated from the pericardium, this structure is removed. When there is only segmental involvement of the diaphragm, a small segment of the muscle is resected with the diaphragmatic pleura. When a large portion of the diaphragm is involved, either by multiple pleural implants or by direct extension, the whole hemidiaphragm should be resected. In four of our patients postoperative chest wall defects have been repaired with stainless steel wire mesh: two of the defects were due to invasion of the chest wall by primary carcinoma of the lung; the remaining two were of localized malignant mesotheliomas.

SUMMARY AND CONCLUSIONS

1. The surgical treatment of a series of twenty-eight cases of serious coexisting pulmonary and pleural disease is described. Pleuropulmonary resection was done in each instance. Subtotal or total hemidiaphragmectomy was performed as adjunctive treatment in eleven instances. Only one postoperative death was encountered; the remaining patients tolerated the surgical regimen.

2. The authors' experience suggests that pleuropulmonary resection and hemidiaphragmectomy may prove of value in the treatment of primary carcinoma of the lung and pleura.

REFERENCES

Davidson, L. R.: Mixed Infection Tuberculous Emphyema, Quart. Bull., Sea View Hosp. **6**: 382-388, 1941.

Hochberg, L. A., Fink, I., and Denize, A.: Revision Thoracoplasty; Study of 89 Cases, Quart. Bull., Sea View Hosp. **8**: 205-211, 1946.

Selikoff, I. J.: Diagnosis of Generalized Amyloidosis by Congo Red Test; Definitive Diagnostic Criteria, Am. J. M. Sc. **213**: 719-727, 1947.

Tchertokoff, I. G., and Selikoff, I. J.: Role of Traumatism in Induction of Initial Pneumothorax; Further Studies, Quart. Bull., Sea View Hosp. **9**: 277, 1947.

Lloyd, M. S., and Naclerio, E. A.: Combined Resection of the Lung and Parietal Pleura in the Treatment of Pulmonary Suppuration and Empyema, New York State J. Med. **49**: 1293, 1949.

Sarot, I. A.: Extrapleural Pulmonary Resection (Pleuropneumonectomy), J. Mount Sinai Hosp. **17**: 700, 1951.

Sarot, I. A.: Extrapleural Pneumonectomy and Pleurectomy in Pulmonary Tuberculosis, Thorax **4**: 173-223, 1949.

DISCUSSION

DR. W. EMORY BURNETT, Philadelphia.—I want to congratulate the essayists on the presentation of an inteersting and rather intriguing subject. I would like to discuss the malignant category only, and state some of our experiences with this.

About three years ago we decided to test the verity of the contraindication of pleural invasion to pneumonectomy, in the hope that this extension might be the only site of secondary invasion. The first patient on whom we intended to try this had been discharged during my absence at this meeting, I believe, to a cancer hospital. When I returned and tried to get him back in, we found he had died the day before of atelectasis of the opposite lung, but at post-mortem there was no invasion whatever of the mediastinal glands. The only secondary spread was that of the pleura, so that we were encouraged to hunt out others.

We have attempted total pleurectomy for carcinoma six times with the hope that it might be at least palliative, and the remote possibility that occassionally it might be curative. Our experience has definitely proved that some of these patients have to be abandoned in the middle of the operation because of the extreme extent found beyond the pleura. That happened in three of the six instances in which we tried it. Of the other three in which it was completed, one lived only about three months thereafter and died of cerebral metastasis; the longest one lasted nine months and was able to go back to work, and to do what he greatly desired, that was some hunting and fishing. His course, however, is illustrative of the increased morbidity as well as possible increase in mortality from this type of procedure. Pneumonectomy, total pleurectomy, and hemidiaphragmectomy on the right side were done. On the fifth or sixth day he eviscerated his intestinal contents through the seventh interspace incision. These were replaced and packed below the diaphragm and liver and did not recur, but the wound was rather slow in healing. He was the one, however, who had the best result so far.

I am pleased to note that there have been some patients in this series reported today who have gone longer, and we understand from four months to two years. I would like to know how many were for two years when the essayist returns.

DR. LOUIS R. DAVIDSON, New York.—I am sure you will agree with me when I say such a wholesome and substantial meal as was served us by Dr. Cotton needs no dessert. Because of this fact, it is with great reluctance that I discuss his paper.

About the time that Churchill and Klopstock reported upon lobectomy in pulmonary tuberculosis, it became apparent to thoracic surgeons that many upper lobes could not be resected from the chest wall, unless one was willing to risk spillage into and infection of the intrapleural space. It was natural, therefore, to seek another avenue of approach in those so-called frozen lobes or lungs. For reasons which must be quite clear, surgeons who had previous training in pathology were quick to use, in part at least, what is known in the autopsy room as the Rokitansky technique—that is what is now called extrapleural dissection or mobilization.

At Sea View Hospital, about 400 patients have been submitted to excisional surgery. Of this number, about eighty or more have had extrapleural resectional surgery. I shall confine my discussion to twenty-nine cases of mixed infection tuberculous empyema occurring in a series of 200 consecutive cases of excisional surgery performed between March, 1946, and February, 1949.

Prior to 1946, various surgeons when reporting on mixed infection tuberculous empyema indicated mortality figures in the neighborhood of 50 per cent. Dr. Cotton's figure, you will note, is lower than this. At the time pneumonectomy and pleurectomy were used at Sea View Hospital, in this group of twenty-nine cases the ultimate mortality rate was 10.3 per cent; three patints having died.

Technically, pneumonectomy and pleurectomy are more difficult for the surgeon and immediately more serious for the patient when compared with the previous methods of thoractotomy, multiple-stage thoracoplasty, and Schede. However, with pneumonectomy and pleurectomy, the end results are far superior.

Of the twenty-nine cases of mixed infection tuberculous empyema, ten had bronchopleural fistula demonstrated preoperatively. Of the remaining nineteen cases, we have assumed from a pathologic as well as from a clinical viewpoint that all empyemas of this type at one time had had bronchopleural fistula.

Results.—

A. Twenty-four, or 82.7 per cent, are living and well with negative sputum.
B. Two, or 7.0 per cent, are living and well, but the sputum is positive.
C. Three, or 10.3 per cent, died.

Of course, pneumonectomy and pleurectomy prolong the operating time and invite a greater loss of blood. However, in another series, we have found that in all types of cases having pneumonectomy and pleurectomy the complications are more frequently noted upon the right side. These difficulties in part are: involvement of the superior vena cava, esophageal fistula, trauma to the diaphragm, etc.

When indications are followed, pneumonectomy and pleurectomy should become a well-established procedure yielding exceptional results.

I enjoyed Dr. Cotton's presentation very much.

DR. DWIGHT E. HARKEN, Boston.—I should like to confine myself to this section of the paper that involves what we, in our Clinic, would regard as meddlesome surgery for obviously hopelessly inoperable carcinoma of the lung. Our tendency has been to do less and less rather than more and more; in other words, to try to make a cleaner, clearer distinction between inoperability and operability and to work toward a more specific procedure consistent with a cancer operation. We have explored the neck and removed lymph nodes routinely in an effort to establish inoperability before proceeding with surgery. This neck dissection is also useful as a diagnostic maneuver in some instances where a diagnosis of cancer cannot otherwise be made. In such a situation it not only establishes the diagnosis but the inoperability thereof. We have, I believe, by this dissection of the neck spared some 50 per cent of our patients surgery that was otherwise nonproductive. We can be guilty of errors of commission as well as errors of omission. The excision of malignant tumors is not always "palliative."

DR. IRVING A. SAROT, New York.—The surgical principle of complete excision has been achieved by pleuropneumonectomy in cases of combined pleuropulmonary disease in tuberculosis, nontuberculous suppuration, and malignancy with secondary suppuration and empyema. Dr. Cotton is to be congratulated on extending the applicability of the procedure by demonstrating the feasibility of adjunctive hemidiaphragmectomy.

Inasmuch as total pneumonectomy and pleurectomy have been discussed here and elsewhere, I should like to confine my discussion to the treatment of pleuropulmonary disease in which only part of the lung is involved; that is, to the development of subtotal resection of the lung with pleurectomy and excision of chronic empyema.

(Slide.) I have found that even the largest empyemas, which may extend over the diaphragm and pericardium, the mediastinal pleura about the hilus, and the subjacent visceral pleura are not usually involved to any great degree by the inflammatory process, as shown in these specimens. This knowledge has been utilized in the development of the extrapleural mediastinal approach which facilitates pleuropulmonary resection.

(Slide.) This sketch illustrates the extrapleural approach to the uninvolved mediastinal aspect, or in less extensive localized empyemas, to the less thickened parietal pleura a short distance away from the empyema. When the thin, relatively uninvolved mediastinal pleura is incised, the visceral pleura is exposed and the proper cleavage plane is entered in which to separate the lung from the overlying thickened parietal pleura and the visceral wall of the empyema. In this manner a lobe of the whole lung may be of the empyema. In this manner a lobe of the whole lung may be freed (decorticated) from the empyema, palpated, and inflated to add information to the preoperative clinical and radiologic evidence upon which to decide whether a segment, a lobe, or the entire lung

must be resected with the empyema. While still working in a clean field, one may then proceed with segmental resection, lobectomy, or pneumonectomy, as required, with the pleurectomy.

(Slide.) This is the specimen in a case in which an upper lobectomy and pleurectomy were performed, and shows the chronic empyema and the open bronchopleural fistula.

(Slide.) The next case is that of a young man with repeatedly negative sputum and gastric contents by all types of bacteriologic examination who, two years after development of a bronchopleural fistula following pneumonolysis, had a large residual tuberculous empyema with fever and malaise.

(Slide.) At exploration through the extrapleural mediastinal approach, the left lung was found to be uninvolved except for the upper lobe apical segment, which was shrunken and indurated. The uninvolved portions of the lung stripped with ease from the visceral wall of the empyema despite its two-year duration. Whether or not a lung is adherent to the visceral empyema wall is dependent on the presence and severity of disease in the lung. Uninvolved lung is usually not firmly adherent, and strips with ease. If the lung is healthy the visceral pleura is uninvolved, and I have been unable to find evidence of fibrosis and ingrowing trabeculae of fibrous tissue from the overlying empyema.

(Slide.) The apical segment was resected with the empyema, and when opened was found to contain a blocked cavity filled with caseous tuberculous material.

(Slide.) Several months later a small apical thoracoplasty closed the residual space, and almost the entire left lung was conserved and is healthy and functioning. This illustrates what may be accomplished by pleuropneumonectomy and its variations, in extirpating the pulmonary and pleural disease while permitting conservation of uninvolved lung in contrast to thoracoplasty and Schede resection of parietes which even at best cause unnecessary collapse and defunctionalization of uninvolved lung.

(Slide.) In some cases, as in this young girl, localized chronic empyemas, without significant associated active tuberculous or suppurative disease in the lung, may be resected without removing any lung. The intact excised empyema sac is shown. Such excision of chronic empyema removes the disease at once, conserves the lung function, precludes long periods of drainage and multiple operations, and obviates the need for thoracoplasty, since the underlying healthy lung can usually expand to fill the space.

(Slide.) This table presents part of my experience with pleuropulmonary resection for tuberculosis and empyema. This group of cases was presented last year at the National Trudeau Society, and I am presenting the same series now, omitting my more recent cases, so that each patient has been followed at least one and one-half years since operation. There were eighteen pleuropneumonectomies for destroyed lung with empyema, eight had obvious bronchopleural fistulas, and seven had thoracotomy or chest-wall sinus openings at the time of operation. Two patients had died when this series was presented last year and another has died since—the mortality, therefore, is 17 per cent, and 83 per cent are alive. One developed a chest-wall sinus, which has recently been excised. The others are well. Pleurectomy for empyema was associated with upper lobectomy in two cases, with upper lobectomy and lower lobe apical segmental resection in three cases, and with upper lobe apical segmental resection alone in one case. All six patients are alive and well. Of the total of twenty-four cases, three have died and twenty-one are alive; the recovery rate is 87.5 per cent, in contrast to the results of treatment by thoracoplasty and Schede. In addition, in one case, a chronic tuberculous empyema was excised without any pulmonary resection and this patient is well.

DR. COTTON (closing).—I wish to reiterate that this paper is a preliminary report. In answer to Dr. Burnett's question, the survival rate for the malignant cases is four months to two years; the survival rate for the inflammatory cases is six months to four years. It was necessary to give up on many of the malignant cases. Fortunately, we have not had Dr. Burnett's experience in the one case of wound disruption. May I thank the discussants for their comments.

THE MANAGEMENT OF THE PATIENT WITH INOPERABLE CANCER OF THE CHEST

RICHARD H. MEADE, JR., M.D., AND RICHARD A. RASMUSSEN, M.D.
GRAND RAPIDS, MICH.

THE cure of the patient is obviously our chief aim in the treatment of any disease. This is no less true in cancer of the chest. However, it is common experience that the majority of patients with this disease are beyond the reach of our present methods of curative treatment when first seen by the surgeon. In 1948, Churchill,[1] in his excellent article on cancer of the lung, quoted the results reported by nine authors after 1940, in which 1,950 cases of cancer of the lung had been seen. In only 22 per cent were resections possible. More recent figures from various parts of the world show very little change (Table I), and our own experience in an area new to thoracic surgery has been

TABLE I. OPERABILITY OF CANCER OF THE LUNG

AUTHOR	TOTAL CASES	RESECTIONS	PER CENT
Mason* England (1949)	1,000	202	20.2
Gagnon† Toronto (1934-1945)	224	49	21.8
Overholt‡ (1932-1949)	721	200	27.7
Burnett§ (1936-1948)	527	94	17.8
Graham‖ (1945-1947)	211	58	27.0
Meade and Rasmussen (1947-1951)	116	27	23.3

*Mason, G. A.: Cancer of the Lung; review of 1,000 Cases, Lancet 2: 587-591, 1949.

†Gagnon, E. D.: Bronchogenic Carcinoma (12 Year Review and Operative Results), Canad. M. A. J. 58: 25-29, 1948.

‡Overholt, R. H., and Schmidt, I. C.: The Silent Phase of Cancer of the Lung. J. A. M. A. 141: 817-821, 1949.

§Burnett, W. E., Rosemond, G. P., Hall, J. H., and Caswell, T.: Carcinoma of the Lung, Arch. Surg. 61: 1052-1057, 1950.

‖Graham, E. A.: Considerations of Bronchiogenic Carcinoma, Ann. Surg. 132: 176-188, 1950.

similar. We have been able to do resections in 23.3 per cent of 116 patients. When we turn to cancer of the esophagus we find it impossible to discover the present operability rate. Much has been written in recent years about cancer of the esophagus, and operability figures have been given, but they are only in terms of the total number of operations and ignore the inoperable cases. The extremely low rate of operability found in 1942 by Santy, Ballivert, and Berard[2] in France, six of a total of seventy patients, is in striking contrast with the present report of Sweet's[3] experience with his private cases since 1944. He has seen 142 patients with cancer of the esophagus and has been able to do resections in ninety-two of them (65 per cent), but he adds that many of these resections were purely palliative. In our much more limited ex-

Read before the Thirty-first Annual Meeting of the American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

perience, we have been able to do resections in nine of twenty-eight patients (32.1 per cent), seen in the last three years. We believe that this more clearly represents the true figure, as we are much more likely to see the hopeless cases than is Sweet. When one adds to these sites the mediastinum and chest wall, no improvement in operability is found. Thus, it is evident that in the majority of our cancer cases the problem is one of the management of the patient for whom no curative surgery is available. And again according to Churchill,[1] if we are to maintain our positions as doctors rather than as artisans, the responsibilities for the care of these patients must be faced. It is the purpose of this presentation to consider the basic principles involved, to cite three unusual cases of inoperable cancer in which unexpectedly good results were obtained, and to outline a plan for the management of these unfortunate patients.

Fig. 1.—Esophagram of patient, Case 1, in February, 1948, showing narrowing of lower third of the esophagus.

There are many in this audience who can well remember when diabetes and pernicious anemia were in the same class with inoperable cancer today. The dramatic change in the outlook for diabetics following the discovery of insulin will never be forgotten by those who first had the opportunity of using this agent. Nor will it be hard to remember the introduction of liver therapy for pernicious anemia. Since then we have seen the great results obtained from the use of the sulfonamides, and the antibiotics, and the amazing changes produced by the use of cortisone and ACTH. How can one doubt the possibility of discovering some agent which will be effective in the treatment of

cancer? It may not come in our lifetime, but we believe that it is of the utmost importance to impress upon the patient with inoperable cancer that such a discovery is a real possibility, and that it might come in time to help him. If we cannot cure our patients, we must not rob them of hope.

In the management of patients with cancer of the chest, the first step is of great importance. We must prepare the patient to accept whatever treatment is finally given as the best in his case. If he is told that he can be cured by the removal of his lung, and he later learns that his lung was not removed, he is left with no alternative but to believe that his case is hopeless. He must be told that while surgical removal is the quickest method of cure for the suitable case, there are certain types of cancer that can best be treated by other methods, and that the surgeon can only make the decision as to the best plan of treatment after exploring the cancer at the operation.

Fig. 2.—Biopsy of esophageal tumor in Case 1 showing a typical squamous-cell carcinoma.

While it is known that the duration of life with inoperable cancer is rarely as long as two years, everyone knows of a case in which all predictions for early death were proved false. There are occasional patients which, either without any specific treatment or after roentgen therapy, nitrogen mustard or other agents, live comfortably for years. It is helpful to tell patients with inoperable cancer about such cases, especially when they live in the same community, or are known to the patient. We have had a number of such experiences, and are briefly reporting here the stories of three of them.

The most striking example is the case of a 57-year-old man who was operated upon two years ago because of a carcinoma of the esophagus. The roentgenogram was typical (Fig. 1), and the biopsy (Fig. 2) showed a squamous-cell carcinoma. At operation the lesion was found to be resectable, but there were metastases in his liver and a hard mass in the pylorus. It was

thought unwise to do a resection. A four-day course of nitrogen mustard* was given, and at six- to eight-week intervals his esophagus was dilated over a swallowed string by means of a Plummer bougie, making it possible for him to eat and to return to his former work. Once, ten weeks after operation, the string would not pass and it was necessary to pass it through an esophagoscope. His general condition has been excellent. Recent examination revealed a normal-looking man. The roentgenogram (Fig. 3) shows no evidence of the former esophageal lesion, and on esophagoscopy there was moderate narrowing but no tumor could be seen, and biopsies from the area showed no evidence of tumor.

Fig. 3.—Esophagram of patient, Case 1, March, 1951, showing evidence of return to nearly normal appearance.

The second patient was a woman 82 years of age who, three years ago, was found to have a carcinoma of the upper esophagus. This involved the pyriform sinuses so that an esophagoscope could not be passed. A biopsy showed squamous-cell carcinoma (Fig. 4). The roentgenogram (Fig. 5) showed the obstruction and marked widening of the esophagotracheal space. A gastrostomy was done, and she was started on Teropterin and roentgen therapy.† The latter was soon discontinued because of edema. Two days

*Nitrogen mustard dosage was 0.1 mg. per kilogram of body weight, given intravenously each day.

†Roentgen therapy for Case 2 amounted to 6,400 roentgen units through three portals.

later she was started on a four-day course of nitrogen mustard. Within two days she could swallow, and from then on her swallowing gradually returned to normal, although she had to chew her food well. Esophagoscopy three years later revealed marked narrowing so that the instrument could only be passed into

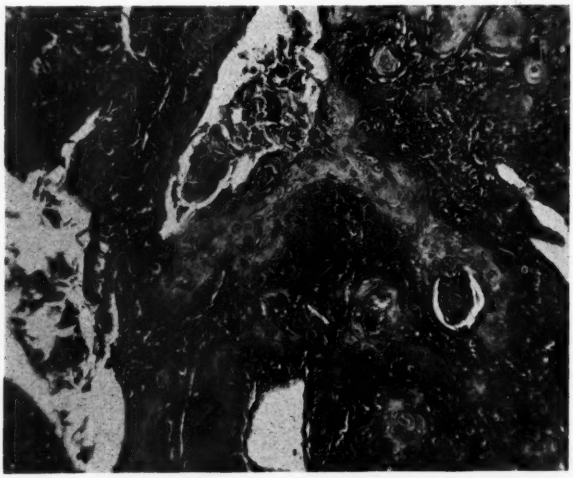


Fig. 4.—Biopsy of tumor filling upper esophagus of patient, Case 2, showing typical squamous-cell carcinoma.

Fig. 5.—Roentgenogram in Case 2, showing marked deformity of upper esophagus and widening of the tracheoesophageal space.

the upper inch of esophagus, but the mucosa appeared normal. This was thought to be due to periesophageal fibrosis due to roentgen therapy. Fluoroscopic examination revealed no abnormality, but the barium passed too rapidly to allow good films to be taken.

The third patient was a 60-year-old man who had a cough and some dyspnea. Roentgenograms (Figs. 6, 7, and 8) showed evidence of marked pressure on the anterior tracheal wall, and bronchoscopy revealed the trachea to be flattened to a narrow transverse slit with roughening and induration of the mucosa and a broad carina. No biopsy was taken for fear of producing tracheal obstruction. He was told that he had cancer which could not be treated surgically, but that he would be given nitrogen mustard. As he knew

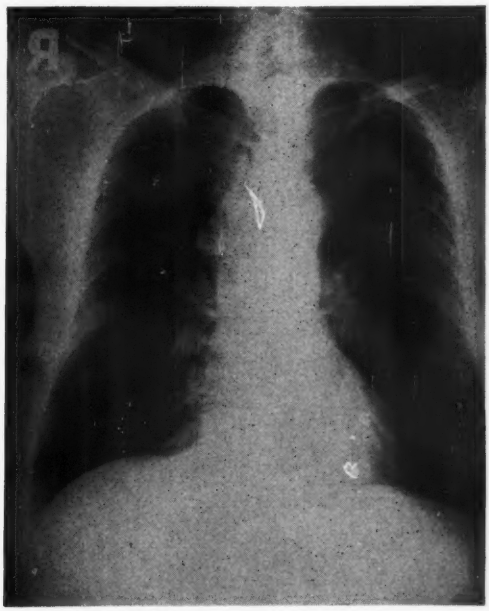


Fig. 6.—Posteroanterior roentgenogram of the chest of patient, Case 3, showing no obvious abnormality.

the patient with cancer of the esophagus who had apparently been so dramatically helped by nitrogen mustard, he was strongly reminded of her case. He received a four-day course of nitrogen mustard without noticeable effect. Later he was given a second course of nitrogen mustard in preparation for roentgen therapy, as it had been pointed out by Karnofsky[4] of the Memorial Hospital, New York, that this would prevent the development of edema. Roentgen therapy was started two days later and continued without incident until he had received 10,000 roentgen units. He improved greatly and carried on his work. Later an x-ray film showed a nearly normal trachea (Fig. 9) and a negative routine chest film. One month later he developed slight ankle edema and

recurrence of dyspnea. On fluoroscopy the heart was markedly enlarged, and he was admitted to the hospital, where he died ten days later. Autopsy showed extension of his carcinoma downward into the mediastinum and into the heart. The trachea, however, was not narrowed.

It is not the object of this article to discuss the value of the forms of treatment used in the three cases just presented, as it is realized that many others have had similar experiences. However, we believe that certain features are worth emphasizing. The first is the use of nitrogen mustard immediately to precede roentgen therapy in patients who might suffer from the development of edema. This effect was dramatically illustrated in our third case where

Fig. 7.—Lateral roentgenogram of patient, Case 3, showing evidence of tumor causing marked pressure on anterior surface of the trachea.

edema might have proved fatal. The other form of therapy which we believe worth stressing is the use of dilatation of the esophagus in patients with inoperable cancer. The passage of a Plummer bougie over a previously swallowed string rarely causes any trouble, and makes it possible for many of these patients to continue to swallow food up to the time of their death. Vinson,[5] and other bronchoesophagologists, have stressed the great value of this procedure for many years, but it is rarely even mentioned in articles by others. In our limited experience we have been greatly impressed with its value.

All of these patients were told that they had carcinoma. They were told that their carcinomas could not be treated surgically but that other forms of

Fig. 8.—Lateral tomogram of patient, Case 3, showing evidence of tumor, causing marked pressure on the anterior surface of the trachea.

Fig. 9.—Lateral tomogram of patient, Case 3, following nitrogen mustard and roentgen therapy, showing nearly normal tracheal lumen.

treatment would help them, and that they had to hope that they might be cured. The one patient who has died was able to lead a normal life up to ten days before his death, even though he lived only four months after the tumor was first recognized. Two of these three patients are still alive and have no evidence at present of their former carcinoma.

The problem of how much to tell a patient with inoperable cancer is not a simple one, and we do not believe that any rigid rules can be made. There is evidence that cancer patients in general do want to know the truth about themselves. The studies[6] made at the University of Minnesota showed that practically all of the cancer patients questioned there were emphatic in their desire to know the truth about their own conditions, and that they believed the great majority of all cancer patients should be told the truth. However, this study was concerned with the reaction of the patient to the knowledge that he had cancer, not that he had an inoperable tumor. Certainly, the information about inoperability should not be given until the patient has recovered from his operation. To give it earlier might prove to be an unbearable shock. The intelligence of the patient, his financial position and the character of his family are all important factors. To withhold this vital information from the head of a family might prove disastrous to the family. However, we are discussing the subject only from the standpoint of the patient. Will he be happier knowing the full truth? If he has no symptoms, or can explain those he has on the basis of some obvious cause, he will live happily without the knowledge of his condition. If he has symptoms that cannot properly be explained without telling him the truth, he will worry more and more about himself, and become convinced that his doctor does not realize how sick he is. He will lose confidence in his doctor and will find fault with everyone dealing with him. Those who are told the truth, in our experience, have at once changed their attitudes and have spent their last days in peace.

Although the patients upon whom most of us operate are referred by other physicians, and after operation are referred back to them, the responsibility for their fundamental care should remain with the surgeon. He it is who makes the decision as to the definitive treatment. We believe he, rather than the family doctor, should tell the patient of his condition. It is frequently impossible for the surgeon to follow his patients in person, but we believe that his responsibility as a consultant does not end with the patient's discharge from the hospital. The patient with inoperable cancer who is discharged by his surgeon must feel that his death sentence has been sealed. The knowledge that his surgeon is still interested in his case, and is on the alert to bring him any hope, will give him a sense of security and an optimistic outlook that will be of great value.

CONCLUSIONS

Because the great majority of patients with cancer of the chest are not seen early enough to allow for curative surgery, their management is obviously one of the greatest responsibilities of the thoracic surgeon. As the use of various forms of nonsurgical treatment have at times proved of value,

we believe they should be used in most cases of inoperable cancer. Furthermore, we believe that in general all patients with inoperable cancer of the chest should be told the basic truth, but that hope for their recovery must never be destroyed.

REFERENCES

1. Churchill, E. D.: Primary Carcinoma of the Lung, J. A. M. A. 137: 455-461, 1948.
2. Santy, P., Ballivert, M., and Berard, M.: Thoracic Esophagectomy for Cancer, J. THORACIC SURG. 12: 397-431, 1943.
3. Sweet, R. H.: Personal communication.
4. Karnofsky, D. A.: Personal communication.
5. Vinson, P. P.: Diseases of the Esophagus, Springfield, Ill., 1949, Charles C Thomas, Publishers.
6. Kelly, W. D., and Friesen, S. R.: Do Cancer Patients Want to Be Told? Surgery 27: 822-827, 1950.

DISCUSSION

DR. RICHARD H. MEADE, Jr., Grand Rapids, Mich.—It may seem strange to you that at this meeting where we are discussing scientific subjects and discussing definitive treatment of patients, we should introduce the subject of the management of the patient with inoperable cancer. So far as I can remember, the last time any speaker before this organization mentioned the patient as an individual was in 1934, when Rudolph Jessen of Switzerland discussed the treatment of bilateral tuberculosis by means of thoracoplasty. Unfortunately, the discussion of that paper was not published, but those of us who heard it will never forget the beautiful tribute that Dr. Lambert paid to Dr. Jessen. I am sure that all of us are impressed with the large number of patients with cancer for whom we have no definitive treatment. However, we must remember that they are individuals, and that we owe something to them even though we cannot cure them of their disease.

DR. DEAN B. COLE, Richmond, Va.—I am glad to hear this excellent paper, but do not agree that all patients with cancer wish to know or should know the diagnosis. We see a number of patients who probably suspect they have cancer, but never ask.

I think it a mistake for the surgeon to be in too great a hurry to inform the patient of his diagnosis, and I think he should be especially careful if the referring doctor is to take care of the patient during the remainder of his illness. It is easy to be scientifically cold-blooded in passing a death sentence, even though some hope may be held out for a reprieve. I think the family should always know the diagnosis. Every possible consideration should be given the patient in talking to him or his family.

DR. GABRIEL P. SELEY, New York.—I would like to report an experience which we have had with three cases recently, which deals with this subject. The first was a 62-year-old man who was operated upon on the private service of Dr. Aufses. This man had a positive biopsy from the trachea and right main bronchus for squamous-cell carcinoma. At the insistence of the medical man this patient was treated with radiotherapy by the grid technique. Following a course of radiotherapy, bronchoscopy showed the trachea and bronchus both negative. Operation was performed, a total pneumonectomy was done, and at that time a tumor was noted in the lung, but the mediastinum was clear. The pathologic specimen showed a squamous-cell carcinoma of the lung with no involved lymph nodes, and there was still residual tumor in the upper lobe bronchus. This man developed a rather indolent type of postoperative wound infection and empyema, but has done fairly well.

The second patient was a man 65 years of age, who was operated upon on the service of Dr. Touroff. This man had a positive biopsy for carcinoma of the left upper lobe bronchus, small-cell in this case. He had a large mass in the lung and was treated by radiotherapy in the same manner. The lung mass decreased in size. Bronchoscopy showed a negative bronchial biopsy. He was operated upon; a pneumonectomy was performed. At that time it was noted that he had a mass in the lung but the mediastinum was completely free. In this case numerous and careful examinations of the specimen removed have failed to show any evidence of carcinoma. Encouraged by these two results, a third patient was treated in a similar man-

ner. He was a 61-year-old man who had a six-month history of carcinoma of the lung. He had a positive biopsy, both from the carina and the right upper lobe bronchus, for squamous-cell carcinoma. He was given radiotherapy. He was then re-bronchoscoped, and the carina was negative. A pneumonectomy was performed, a right pneumonectomy, at which time a mass in the lung was noted, but the hilus was free. The pathologic specimen showed squamous-cell carcinoma of the lung, but no evidence of endobronchial lesion and no involved lymph nodes.

Both the latter two patients had primary union of their wounds, and both have done well. This, I think, is another method which may be considered, and may be employed in order to make some of these patients who seem to be inoperable, operable.

DR. ROBERT R. SHAW, Dallas, Texas.—In discussing this always timely paper by Dr. Meade and Dr. Rasmussen, I would like to add two instances to the rays of light in the dark corner.

(Slide.) This is a patient whom I saw in October, 1945. I am indebted to the Scott and White Clinic of Temple, Texas, for the first film. This large shadow in what proved to be the anterior portion of the left chest was thought to be a mediastinal dermoid tumor, but on exploration I found that it was a large mass arising in the left upper lobe which had invaded the pericardium. I opened the pericardium and found approximately 300 c.c. of bloody fluid in the pericardial sac. It was more expedient to take the biopsy from within the pericardial sac than at any other part of the tumor mass, so biopsy was taken from that point. The chest was closed. The situation was explained to her husband, that it was inoperable, and that the prognosis was very bad. I did advise, however, that she have deep x-ray therapy. This was given, and in the second film you see the result a little more than five years later. This patient has had absolutely no recurrence of symptoms, and is leading an entirely normal life. The only complaint is that she is a bit overweight.

(Slide.) This is the film of a 54-year-old man whom I saw in December, 1945, now in his sixth year. The bronchoscopy was positive for epidermoid carcinoma, the biopsy being taken from the right upper lobe orifice. I advised exploration, but at exploration I found that the carcinoma, although not large, had extended up the tracheal wall so that I thought it impossible to remove it safely. The phrenic nerve was involved in the tumor, and I cut that because he was having intractable shoulder pain. After exploration, the usual bad prognosis was given to the wife, but I did advise that he have x-ray therapy.

In the next film you see the result two months following a course of x-ray therapy that took approximately three weeks to give.

(Slide.) Here we see the result six months later when the right upper lobe had completely broken down in suppuration. He was a thoroughly miserable man, coughing out purulent sputum that was foul, and running temperature elevation. But gradually he began to improve, and the last film shows the appearance of his chest for the past three years. It has not changed in three years' time. At the time exploration was done he had just been re-elected mayor of a small city in east Texas. I made a mental note that he would not live out his two-year term. He did live out his two-year term, but he was not feeling too well at that time so he did not run for re-election. However, at the last election he did run, and is now mayor of the city and is feeling very well. I am getting quite weary of telling his wife that this man can't live very long.

Of course these are exceptions to what we usually see, but I happen to live in a community where the radiologic pace is set by a man who believes in carrying x-ray therapy to safe limits. In other words, he gives them a good big dose, and the other men working in the community do the same. It is rather strange to have heard the two very fine presentations this morning telling of the extension of the surgical attack upon this disease, and then think of the plea made by the usual surgeon to the radiologist, not to make these patients too sick with their x-ray therapy, "and please don't burn the skin, because we just want to palliate the cough a little bit." Sometimes I believe if x-ray therapy is carried to its logical conclusion, an amazing result may be obtained.

DR. PAUL HOLINGER, Chicago.—This excellent paper by Dr. Meade and Dr. Rasmussen certainly raises some fundamental points and indicates, in a way, a justification for a subspecialty of broncho-esophagology. The surgeon doing endoscopic work is primarily interested in the diagnostic phases of the field; the endoscopic management of the patient requiring a nonsurgical course of treatment frequently reverts to the otolaryngologist who is doing endoscopic procedures, to the broncho-esophagologist. This was emphasized in Dr. Olsen's discussion, when Dr. Tuttle mentioned that surgery may be necessary in benign stenotic esophageal lesions because one cannot get anybody to dilate them. Dr. Olsen, you recall, answered that very well by simply stating that for a man who could take out the auricles of the heart, dilatation of the esophagus should not be too difficult.

Dr. Meade has stressed the fact that patients with inoperable esophageal carcinoma can be treated palliatively by dilatation and by roentgen therapy. We have used a resectoscope technique using a cystoscopic type of resectoscope to resect the carcinomatous tissue when the lesion is inoperable, thus permitting the patient to swallow in a normal manner. In inoperable carcinoma of the tracheobronchial tree the removal of tumor tissue from the trachea with the resectoscope is feasible. Radon seed implantation of tumors likewise should not be overlooked in otherwise inoperable patients. Two- to three-year alleviation of symptoms in patients with inoperable esophageal cancer is not at all uncommon under such management, and in one case such treatment permitted normal swallowing for six years. In the tracheobronchial tree, such management of tracheal tumors is particularly noteworthy. Repeated endoscopic resection of carcinomatous tissue in one inoperable cancer of the trachea was done for seven years, stressing the value of endoscopic management of the patient with inoperable cancer.

The danger of such reports is that the broncho-esophagologist is accused of keeping patients from the surgeon when the lesions are resectible. This is not the case. The broncho-esophagologist is just as desirous to refer the patient for surgical removal of these lesions when such removal is possible, as the surgeon is to see the patient. I think, however, that the management of the inoperable patient which Dr. Meade and Dr. Rasmussen have stresssed, should continue with the endoscopic procedures that have been mentioned.

MR. W. P. CLELAND, London, England.—I have been most interested in this discussion, and have been prompted to tell you a little of my own experiences in the field of carcinoma of the bronchus in connection with x-ray therapy. Three years ago a man, aged 40 years, with carcinoma of the bronchus, came to me, and when I bronchoscoped him I found a tumor arising in the right upper lobe, extending up the right main bronchus practically to the carinal level. There was marked subcarinal widening and I considered that, bronchoscopically, he was inoperable. A biopsy showed an oat-cell carcinoma, and I referred him to an x-ray therapist who gave him a radical course of x-ray therapy spread over a period of about six weeks. I saw him again about four months later, and his improvement was dramatic. The solid upper lobe was completely aerated, he had put on 30 pounds in weight, and when I bronchoscoped him a second time there was no evidence of tumor in the bronchus. I subsequently performed a radical pneumonectomy without any difficulty. The specimen failed to reveal any macroscopic or microscopic evidence of tumor, but two years later the man died from secondary deposits.

This prompted me, about eighteen months ago, at the Postgraduate Medical School in London, to start a clinical experiment combining preoperative x-ray therapy with resection. During the first twelve months of that experiment 175 patients were referred for assessment, and fifty of those were considered to have only a primary lesion without obvious secondary deposits either clinically or with x-rays. The interesting thing about these cases which were all treated radically with x-ray therapy has been the tremendous improvement shown by the majority of these people. The tumor masses in many cases have disappeared, the lung has re-aerated, and improvement in the general condition has been most striking. Twenty-four pneumonectomies have been performed to date on these cases, and the operative difficulties were not any greater than in the untreated cases.

I would like now to mention a small group of twelve cases who were regarded, before x-ray therapy, as being bronchoscopically inoperable. That means either that the growth was too close to the trachea, or there was widening below the carina, to justify a pneumonectomy. Five of those patients subsequently became resectable, and pneumonectomies were performed. Their subsequent course has not been very different from the normal pneumonectomy, although one patient has since died from a recurrence in the stump. It is far too soon to decide whether this combined treatment is going to be an advance with a longer survival rate, but I think the experiment is worth continuing.

I do not think any mention has been made this morning of the palliation of carcinoma of the bronchus, or treating superior mediastinal obstruction or secondary deposits in bone; both of the latter respond extraordinarily well to x-ray therapy, with considerable symptomatic improvement, even though it is temporary.

DR. RASMUSSEN (closing).—We wish to thank the discussers. With regard to Dr. Cole's remark, we did not mean to imply that he too should not talk to his patient about this terrible condition, because he undoubtedly will have more contact with him, but it is sometimes helpful if both physician and surgeon talk to the patient.

It is probably unwise to make predictions as to the time of death lest the radiologist or someone else afford the patient the advantage of an unusual outcome.

RADIOACTIVE ISOTOPES IN THE PALLIATIVE MANAGEMENT OF CARCINOMATOSIS OF THE PLEURA

EDWARD M. KENT, M.D., AND CAMPBELL MOSES, M.D. (BY INVITATION)
PITTSBURGH, PA.

CARCINOMATOSIS of the pleura associated with a profuse effusion represents one of the complications of malignant neoplasms which is encountered with some frequency. The clinical management of such complications has always been difficult and unrewarding if the effusion formed rapidly. Victims of this unfortunate situation suffer from recurring, severe dyspnea as the pleural space inexorably fills with fluid after each aspiration. The loss of large quantities of protein into the effusion increases the wasting and hastens death materially. Few individuals live more than weeks once carcinomatosis of the pleura becomes established, provided an intractable, rapidly reforming effusion is also present.

The treatment of such effusions has been ineffective in the past. In our experience efforts to stop or delay the formation of pleural fluid have been to little avail. Roentgen therapy has offered the most benefit, but failure has been experienced in the great majority of patients given irradiation therapy by this means. Some reasons for this failure are obvious. One cannot irradiate adequately the entire hemithorax of an individual, nor can the rays be directed accurately at the areas of pleural implantation, since their precise location is unknown and they are usually numerous and scattered widely on both surfaces of the pleural cavity.

Radioactive isotopes were considered as a possible source of irradiation therapy under such circumstances, inasmuch as these substances can be injected directly into the pleural effusion and thereby irradiate all surfaces of the cavity. Because of the relatively short range of beta rays in tissue (0.8 to 2 mm. for the beta rays from I^{131}), it was thought that relatively large doses of radiation could be delivered to the pleural surface with a minimum of radiation damage to adjacent structures.

At the time this study was begun, no data were available on the absorption, excretion, stability, or toxicity of radioactive material administered by the intrapleural route. However, previous experience by several groups of workers using large doses of I^{131} in the treatment of thyroid carcinoma had established the safety of this procedure both to the patient and attending personnel. For these reasons, and because of its ready availability, carrier free I^{131} in weak basic sodium sulfite solution as supplied by the Oak Ridge National Laboratory was initially used in this study. As this work progressed, radioactive colloidal

From the Thoracic Surgery Division, Department of Surgery, and the Addison H. Gibson Laboratory of the School of Medicine, University of Pittsburgh.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, N. J., April 16-18, 1951.

gold (Au^{198}) and I^{131} tagged human albumin became available* for investigational use and these isotopes were used in a limited number of patients in this series.

MATERIAL AND METHODS

Only patients with neoplastic disease and established carcinomatosis of the pleura who demonstrated a rapidly forming, intractable pleural effusion were employed for these preliminary observations. In most instances (14) the neoplasm was of bronchogenic origin, in some (4) the primary tumor had been carcinoma of the breast, and in one patient the primary neoplasm was a fibrosarcoma of the abdominal wall which had grown to enormous size over a period of seventeen years and had resulted in extensive intrathoracic metastases with intractable pleural effusion.

An arbitrary dosage of 10 millicuries of I^{131} was chosen initially for this investigation, but this dosage was later increased to as much as 30 millicuries. The I^{131} as obtained from Oak Ridge was transferred to a hot, sterile screw cap bottle of 50 to 100 ml. capacity. This preparation was found to be bacteriologically sterile. Under local anesthesia the pleural effusion was aspirated as completely as possible. During the aspiration the I^{131} containing bottle was filled with pleural fluid. At the conclusion of the aspiration the I^{131} containing pleural fluid was reinjected into the pleural cavity. This fluid was aspirated and reinjected several times in an attempt to deliver all of the isotope into the pleural space.

Immediately after completing the injection the operator thoroughly rinsed the gloves, syringe, needle, tubing, and stopcock in cold running water. If this washing is promptly and thoroughly done no significant contamination can be detected on the equipment.

In most of the patients here reported the urinary excretion of isotope in the two to five days following its administration was determined. Details of this excretion study will be provided in a separate report. However, of the twelve patients receiving I^{131} in this series on whom excretion data was obtained, 62 per cent of the administered I^{131} was retained forty-eight hours. Although accurate quantitative determinations of thyroid uptake were not made, undoubtedly much of this activity was retained in this gland after removal from the pleural space. In Case 10, five days after the administration of 25 mc. of I^{131}, only eight per cent of the administered radioactivity remained in the pleural fluid. When this same subject was given 5 mc. of I^{131} tagged albumin 93 per cent was retained in the body five days after administration and 72 per cent was retained in the pleural fluid. A similar study with radioactive colloidal gold in Case 11 revealed only 0.47 per cent of the gold appearing in the urine in seventy-two hours, and 23 per cent was retained in the pleural fluid on the fifth day.

RESULTS AND CASE REPORTS

The results of therapy, the isotopes with the doses employed, and the types of carcinoma present are indicated in Tables I and II. Analysis of these tables

*Through the courtesy of D. L. Tabern, Ph.D., Abbott Laboratories, Chicago, Ill.

TABLE I. SUMMARY OF DATA AVAILABLE IN THE STUDY OF FOURTEEN PATIENTS WHOSE PRIMARY CARCINOMA AROSE IN THE LUNG

CASE NO.	TYPE OF CARCINOMA	ISOTOPE AND DOSAGE	RESULTS
1	Undifferentiated	10 mc. I^{131}	Fluid checked but observation limited to 21 days by death
2	Epidermoid	10 mc. I^{131}	Materially improved. Aspiration only once in two months of life following treatment
3	Squamous-cell	10 mc. I^{131}	Fluid completely checked. Now alive more than 12 months
4	Undifferentiated	20 mc. I^{131}	Effusion materially retarded—Aspirated twice in 3½ months of life after therapy—total 3100 c.c. Previously aspirated twice weekly average of 2700 c.c. each time
5	Undifferentiated	10 mc. I^{131}	Effusion completely checked. Lived 8 months
6	Squamous-cell	10 mc. I^{131}	Effusion completely checked. Lived 10 months
7	Undifferentiated	30 mc. I^{131}	Died 17 days after therapy. Cannot be evaluated
8	Squamous-cell	37.6 mc. Au^{198}	Effusion checked. Only 6 weeks follow-up
9	Adenocarcinoma	19.2 mc. I^{131}	Died 15 days after therapy. Cannot be evaluated
10	Adenocarcinoma	5 mc. albumin I^{131} 55 mc. I^{131} 120 mc. Au^{198}	Most refractory to treatment of all cases. Effusion very materially slowed. Now 4 months post treatment
11	Undifferentiated	40 mc. Au^{198}	Died 13 days after therapy. Cannot be evaluated
12	Squamous-cell	2.475 mc. I^{131} in serum albumin	Effusion completely checked. Alive 6½ months
13	Squamous-cell	20 mc. I^{131}	Effusion completely checked. Died 2 months
14	Undifferentiated	20 mc. I^{131}	Effusion completely checked. Alive 4 months

TABLE II. SUMMARY OF DATA IN THE STUDY OF FOUR PATIENTS IN WHOM THE PRIMARY CARCINOMA AROSE IN A BREAST AND OF ONE PATIENT WHOSE PRIMARY TUMOR WAS A MYXOFIBROSARCOMA OF THE ARTERIOR ABDOMINAL WALL

CASE NO.	TYPE OF CARCINOMA BREAST	ISOTOPE AND DOSAGE	RESULTS
15	Adenocarcinoma	10 mc. I^{131}	Completely checked effusion. Died in 4 months
16	Adenocarcinoma	20 mc. I^{131} each pleural cavity	Bilateral pleural effusion. No further aspirations required after therapy but pleural spaces were not dry. Died 3½ months after therapy
17	Adenocarcinoma	30 mc. I^{131}	Completely checked effusion. Alive 2 months
18	Adenocarcinoma	18.58 mc. I^{131}	No further aspirations required. Pleural cavity not dry. Died 4 months after therapy
19	Myxo-fibrosarcoma	10 mc. I^{131}	Completely checked effusion. See case report. Died 6½ months after therapy

shows that of the fourteen patients shown in Table I, three died too soon after therapy to be useful in evaluating results. However, in no instance was the pleural effusion a factor in the death of the patient, and the effusion in each case appeared to be checked or greatly retarded. In three instances, the results would be classified as fair since fluid did reaccumulate in sufficient quantity to require aspiration at least once after the immediate post-therapy interval. In eight patients, the effusion was promptly and completely checked. The results shown in Table II are those obtained in four patients, all women, who had the primary carcinoma in a breast, and in one patient whose primary lesion was a myxofibrosarcoma of the anterior abdominal wall. The results were good in each instance, although two of the patients continued to have small amounts of pleural fluid until death.

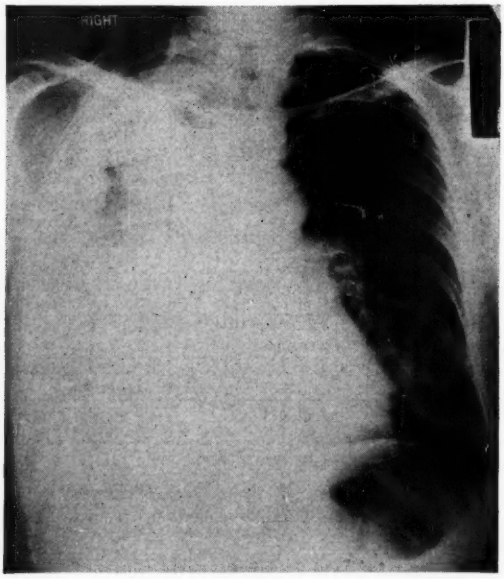


Fig. 1.—Chest film demonstrating right pleural effusion in patient designated in text as Case 1.

A brief description of the first patient treated by this technique in this investigation is presented: J. W., a white man 62 years of age, was admitted to the hospital complaining of severe dyspnea. The clinical findings were those of right-sided pleural effusion, and these were confirmed by a roentgenogram of the chest (Fig. 1). Thoracentesis yielded 500 c.c. of bloody pleural fluid, and at the conclusion of the aspiration the pleural space had not been emptied. Reaccumulation of the pleural fluid was so rapid that aspiration was required at frequent intervals and 4,230 c.c. were removed over a period of seven days. Carcinoma cells were found to be present in the pleural fluid (Fig. 2), and upon bronchoscopy a neoplasm was seen in the right intermediate bronchus (Fig. 3). Biopsy of this lesion revealed an undifferentiated carcinoma. On the fourteenth

hospital day a single dose of 10 mc. of radioactive iodine was injected into the right pleural cavity, and studies of the rate of excretion of the isotope revealed that 64 per cent had been excreted in the urine during the first forty-eight hours immediately following injection. While our first reaction to this observation was one of disappointment and belief that the isotope had been excreted too quickly to have been effective, such was not the case. The patient did not again present signs and symptoms indicating a need for thoracentesis. However, numerous subsequent aspirations were performed for study purposes. During

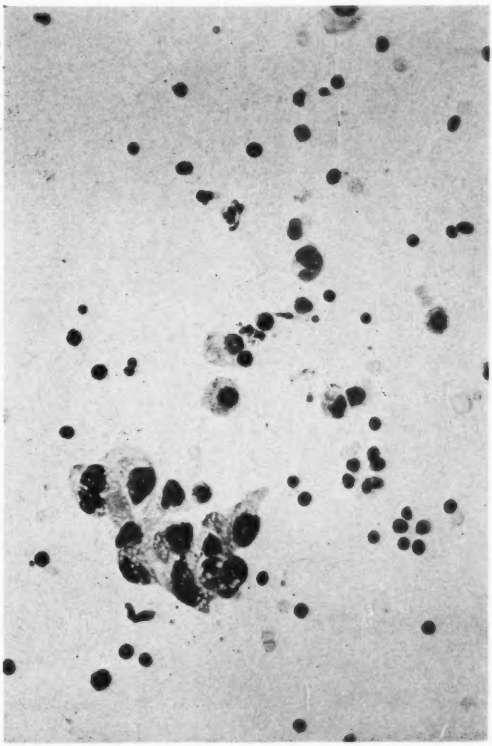


Fig. 2.—Carcinoma cells in the pleural fluid, Case 1. (×540.)

the first five days after treatment 4,450 c.c. of blood-tinged fluid were removed from the pleural space, and on the succeeding days until death "dry taps" were experienced. Twenty-one days after treatment the patient died, and an autopsy was performed. Although there were extensive metastases to the surfaces of the pleural cavity, only a small volume of fluid was present. Microscopic examination of the metastatic implants on the pleura revealed cytological changes in the superficial layers of carcinoma cells indicative of cell death (Fig. 4). Additional autopsy findings included the presence of extensive metas-

tases to the peribronchial and mediastinal lymph nodes, the periaortic lymph nodes, the diaphragm, the liver, the right adrenal, and to the celiac lymph nodes.

A typical good result can be shown by the presentation of Case 2: A 58-year-old woman who had been the victim of an intractable right pleural effusion was admitted to the hospital with a history of repeated thoracentesis during the five weeks immediately preceding hospitalization. A total of 18,000 c.c. of blood-tinged pleural fluid had been removed, and cytological studies of the fluid

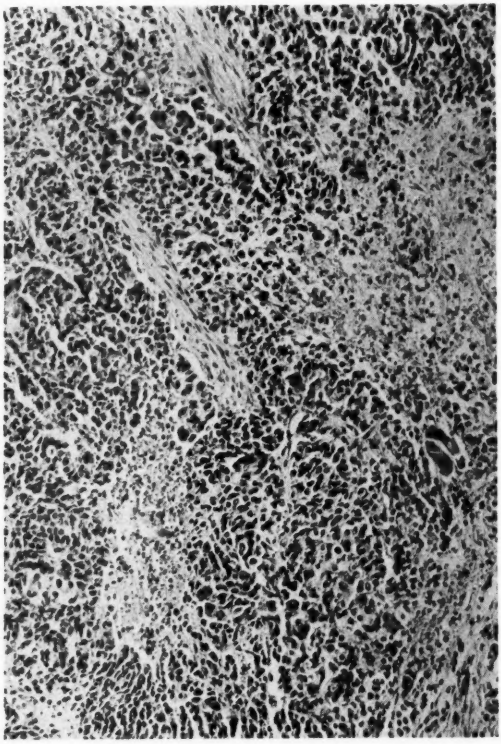
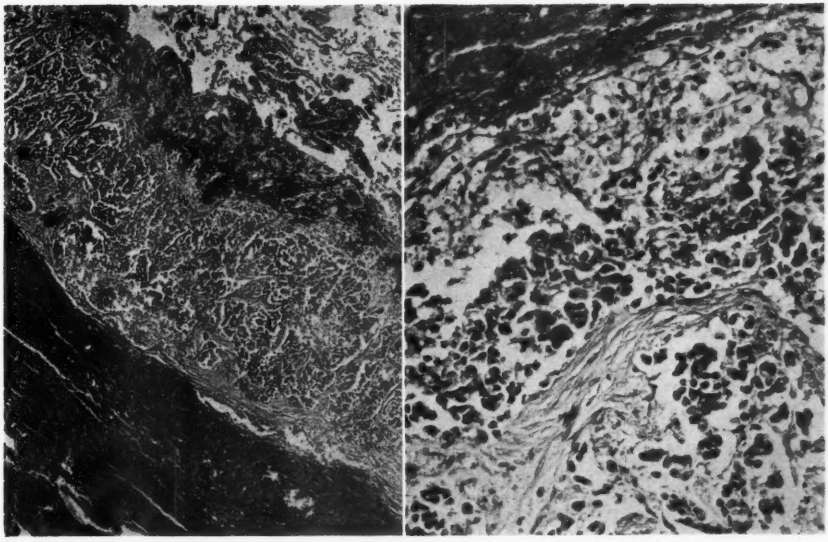


Fig. 3.—Bronchoscopic biopsy from patient in Case 1 revealing undifferentiated carcinoma. (×150.)

revealed carcinoma cells (Fig. 5). Upon admission to the hospital the right pleural space was filled with fluid (Fig. 6), and the patient was suffering pronounced dyspnea and displaying moderate cyanosis. Bronchoscopy revealed a neoplasm which partially obstructed the right lower lobe bronchus, and a biopsy revealed carcinoma (Fig. 7). A single injection of 10 mc. of I^{131} was followed by a cessation of the effusion, and further aspirations were not required. The patient lived for nine and one-half months, and autopsy confirmed the diagnosis of bronchogenic carcinoma with metastases to the pleura.



A. *B.*

Fig. 4.—*A.* Photomicrograph of pleural surface specimen removed at autopsy in Case 1. Changes in the surface cells of the carcinomatous implants are visible. (×75.)
B. Higher power photomicrograph of surface cells of carcinomatous implant. (Autopsy specimen, Case 1, ×275.)

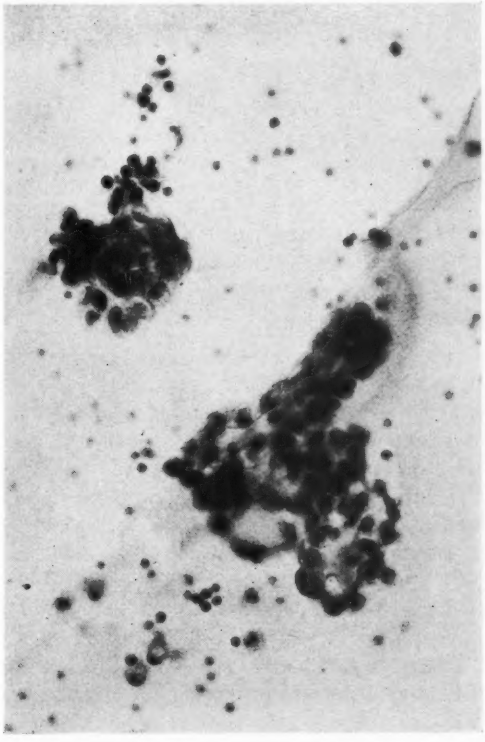


Fig. 5.—Carcinoma cells found in pleural fluid, Case 2. (×540.)

Case 3 is presented because of the unusual history and because of the fact that this patient presented a very large myxofibrosarcoma of the abdominal wall which had metastasized to the lungs and pleura. The patient, a 66-year-old man, was admitted to the hospital complaining of severe dyspnea and orthopnea. Physical signs and a chest film (Fig. 8) indicated the presence of a massive left pleural effusion and a total of 7,000 c.c. was aspirated during the first four days of hospitalization. A very large, nodular, firm mass was present on the anterior abdominal wall which was not fixed to the muscles, and which appeared to have arisen in the subcutaneous tissues. The patient first noticed a small mass at this point seventeen years previously, and gave the history that it had grown slowly during the interval to reach great size, but there never had

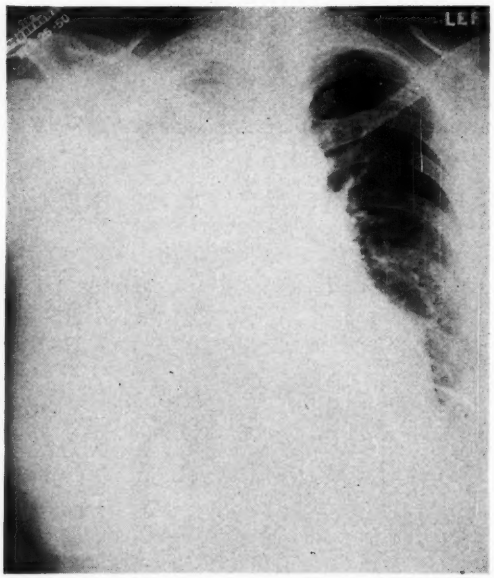


Fig. 6.—Chest film demonstrating massive right pleural effusion, Case 2.

been pain or other discomfort. This mass was removed and was found to be a myxofibrosarcoma of the subcutaneous layer (Fig. 9). In an effort to check the rapidly forming pleural effusion, 10 mc. of I^{131} was injected into the left pleural space. No further aspirations were necessary and the pleural fluid disappeared, revealing a large intrathoracic mass on the roentgenogram (Fig. 10). The patient resumed his occupation and worked for six months. At the end of this period, a rapid decline took place, and death occurred in two weeks. Autopsy findings demonstrated the presence of the massive tumor in the left lung (Fig. 11), multiple smaller metastases in both lungs, and widely disseminated metastases throughout the body. Microscopic examination of these masses (Fig. 12) led to the opinion that they represented metastases from the abdominal

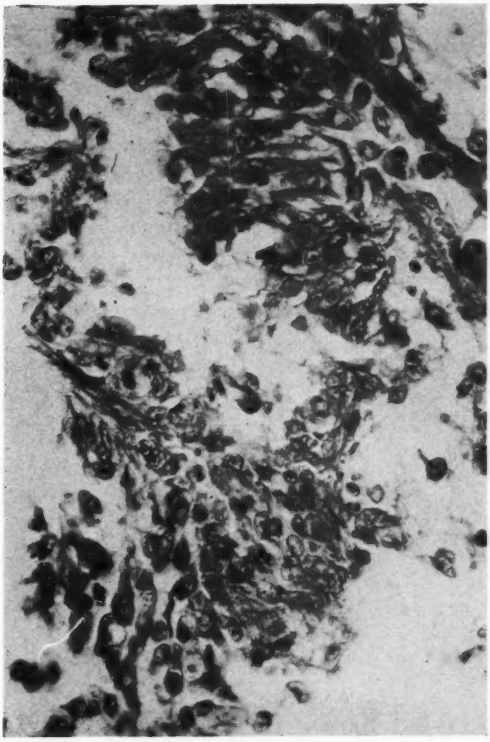


Fig. 7.—Bronchoscopic biopsy demonstrating undifferentiated carcinoma, Case 2. (×540.)

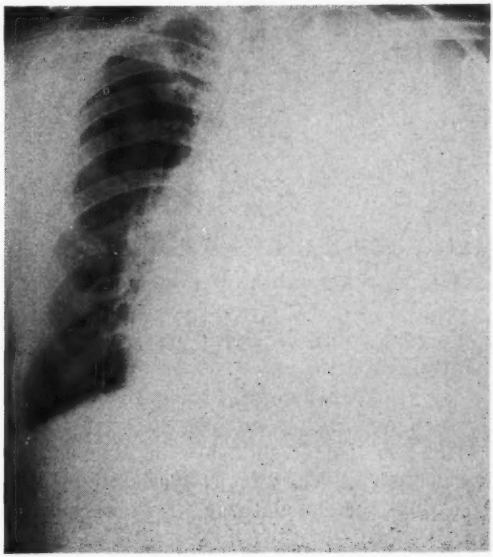


Fig. 8.—Extensive left pleural effusion, Case 3.

sarcoma; however, the cell patterns were not entirely comparable to the findings in the abdominal wall tumor.

The doses of radioisotopes used in this study have not produced evidences of toxicity in any patients. There have been no instances of radiation sickness, and no anemia or leucopenia has been observed to date. While the single dose employed in most patients was not large, one patient in this series (Case 10) was given several treatments and a fairly large amount of radioactivity. This patient was most refractory to this type of therapy. A very profuse, extremely

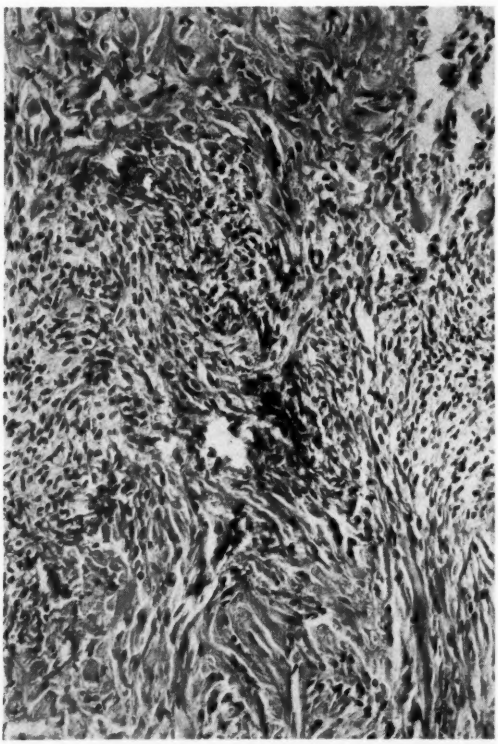


Fig. 9.—Micrographic picture of primary fibrosarcoma of abdominal wall in Case 3. (×135.)

bloody effusion was present which responded very slowly, so that over a period of seven weeks one injection of tagged serum albumin (5 mc.), three injections of I^{131} (totaling 55 mc.) and a single injection of Au^{198} (20 mc.) were given. In the following two months the patient was aspirated on two occasions for a total yield of 1,170 c.c., so that another dose of isotope therapy was decided upon. At this time 100 mc. of Au^{198} was employed, the largest dosage any patient had been given singly or cumulatively. No toxic manifestations have developed in this man.

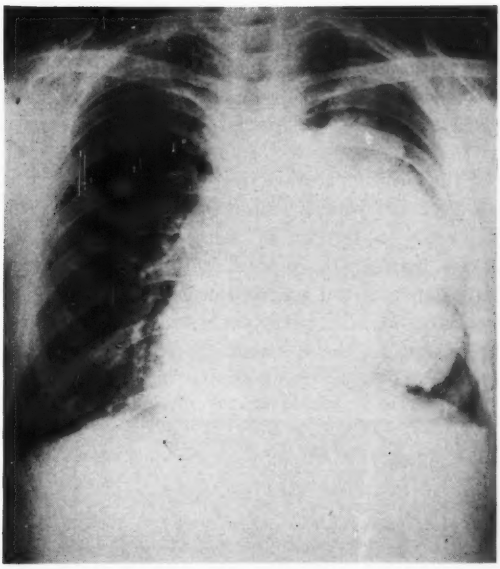


Fig. 10.—X-ray appearance of chest after removal of large amount of pleural fluid Case 3. Enormous left intrathoracic mass visible.

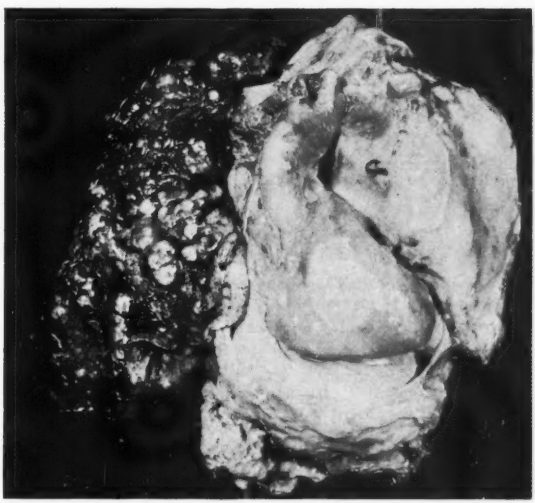


Fig. 11.—Autopsy specimen Case 3, revealing large left pulmonary metastases and numerous smaller right pulmonary metastases.

DISCUSSION

Because of the rapid disappearance of the I^{131} from the pleural space, it is difficult to explain why the beneficial response occurred. It seems unlikely that a beta ray tissue dosage large enough to cause severe destruction of tumor cells was delivered. However, there can be no doubt that fluid production was markedly retarded or stopped. Perhaps this can be accomplished with beta radiation dosage much smaller than that necessary to cause tissue destruction. In Case 9 assay of the residual radioactivity in a sample of pleural tissue taken at autopsy revealed an I^{131} concentration of 0.23 mc. per gram of tissue. Because of the indeterminate location of metastatic implants, it is impossible to calculate the distance between the pleural isotope and the lesion and, therefore, the amount of radiation received by the tissue cannot be reported in roentgens or roentgen equivalents. Additional experimental work is currently in progress in an attempt to determine the optimum radiation dosage and most suitable isotope for this type of therapy.

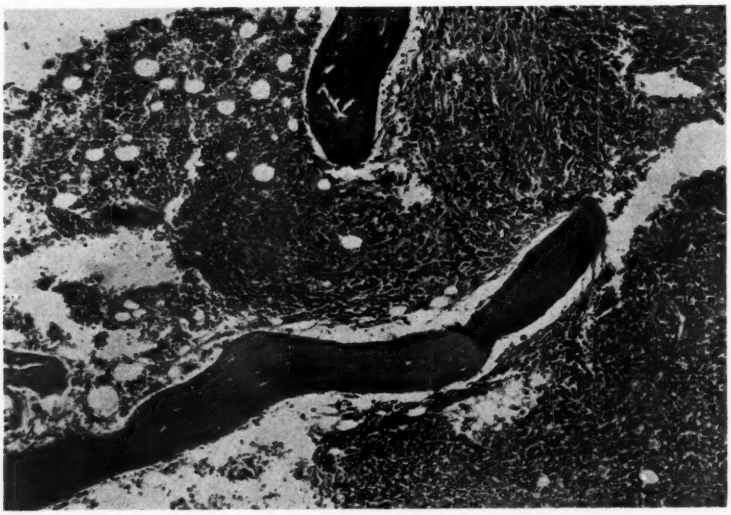


Fig. 12.—Bone marrow metastases found at autopsy, Case 3.

SUMMARY

1. A preliminary report on the first nineteen cases of pleural effusion due to metastatic carcinoma treated by the local injection of radioactive isotopes into the pleural space is presented.

2. Three of these patients died too soon after therapy to evaluate the results, and in three patients the formation of pleural fluid was definitely retarded but not entirely checked. In thirteen patients of this series with metastatic carcinoma the formation of pleural fluid was completely checked by the administration of radioactive iodine, iodine tagged albumin, or radioactive colloidal gold.

3. No toxic manifestations from the administration of the isotopes were noted in this series.

DISCUSSION

DR. J. KARL POPPE, Portland, Ore.—Dr. Kent and Dr. Moses are to be congratulated on this very careful work which represents a great deal of effort I am sure, because Dr. Julius Hessel and I undertook somewhat similar studies with insoluble redioactive ferric phosphate at the University of Oregon Medical School several years ago. This experimental work was confined to dogs and never reached the clinical stage because it seemed impossible to standardize the radioactivity of the ferric phosphate sufficiently to determine its minimal lethal or maximum effective dose. Due to the amphoteric nature of the phosphorus, the colloidal solution of ferric phosphate had a very vague end point on titration. It was very disturbing to discover that up to 50 per cent of the radioactivity of the original phosphorus was being lost in the supranatant fluid rather than being retained in the insoluble ferric phosphate suspension as was desired.

The original purpose in injecting this suspension of insoluble ferric phosphate into the pleural cavity was to irradiate the peribronchial nodes. The ferric phosphate was very easy to follow with iron stains on histologic sections, showing that the ferric phosphate was deposited in the peribronchial nodes. The radioactivity of these nodes was adequately demonstrated by autoradiographs. However, it seemed impossible to obtain a sufficient concentration of radioactivity in the nodes to destroy completely even a normal lymph node, not to speak of a node containing a large area of metastatic carcinoma. The normal morphology and histology of the node would be altered and the germinal centers eliminated, but some lymphatic tissue would always remain intact.

The use of radioactive material injected in the pleural cavity would seem to have definite advantages in maintaining a high concentration of irradiation around the pleural metastases if a completely insoluble substance is used which will not escape into the blood stream, liver, and bone marrow.

DR. RALPH D. ALLEY, New Haven, Conn.—Dr. Kent has mentioned difficulty experienced in the total evacuation of serous effusions by ordinary thoracentesis technique, and I have a technical suggestion to offer based on some experience we have had in our chest service in New Haven.

We have developed an embellishment of the usual thoracentesis technique wherein a No. 14 needle is used in the usual manner through which is threaded a vinyl plastic tubing which just fits inside the diameter of the No. 14 needle, which can then be threaded down into the costophrenic sinus and total evacuation of any serous effusion carried out. I mention this because it may be helpful in his studies.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I think the authors of this paper are to be congratulated for discussion of something which seems to hold a lot of promise. About five years ago, working in Dr. John Lawrence's laboratory, we determined that simple solutions of radioactive phosphorus disappeared from the pleural cavity of dogs in a few hours. About two years ago at The Cedars of Lebanon Hospital, working with Dr. Henry Jaffe in the Department of Radiology, we began to use radioactive phosphorus in simple and colloidal solutions for pleural effusions due to carcinoma. About thirty patients have been treated. The same type of irregular result was noted by us as mentioned by Dr. Kent. Some patients seemed to get a marked dramatic response; others got no response. We then began to use radioactive gold (Au^{198}) for not only pleural effusion but in postoperative patients. So far, six such patients have been treated. One patient developed a bronchopleural fistula about four weeks after instillation of radioactive gold, and that was satisfactorily drained. Radioactive gold does not leave the pleural cavity as readily as does radioactive phosphorus. Also, it does not show itself in the field only. The radioactive energy is far more intense. Instead of Beta rays only, Gamma rays are emitted. The tissues take up the colloidal solu-

tion and fix it, so that although the liver is one of the points where fixation occurs, the tissues of the pleural cavity and mediastinum seem to be also a strong point of fixation of the colloidal gold.

As noted by Dr. Kent, very little is excreted in the urine. It seemed to us that this method might be useful in treating the patient in whom you have left behind some tumor in the mediastinum or pleura following resection. We encountered the same difficulties in calculating the tumor dose as mentioned by other speakers. The dosage usually was 22 mc. in about 30 c.c. of solution of the radioactive gold. In one patient with effusion with an inoperable carcinoma, we put 50 mc. of radioactive gold in the pleural cavity. So far we have seen no effect radiologically after three months, but the tendency to formation of fluid has been lost. This patient developed bone marrow depression; this may be seen at about six weeks after intrapleural instillation.

DR. MOSES (closing).—Dr. Kent and I would like to thank Dr. Poppe and Dr. Alley for their comments, and Dr. Alley for his suggestion as to methods of evacuating the pleura more completely. We would like to emphasize that we do not know why these isotopes exert this effect on the pleural effusion. Certainly the radiation effects are not sufficient to destroy the metastatic lesions. We raise the question that perhaps tissue dosages that are inadequate to destroy the carcinomatous implants but still exert some effect on them may cause sufficient damage to prevent the recurrent formation of pleural fluid.

As to the choice of isotope, we are quite certain now that although most of our cases receive ordinary I^{131}, this is not the optimum form of therapy, and we are quite enthusiastic about the value of colloidal gold and also of iodinated serum albumin. The colloidal gold, as Dr. Goldman indicated, is picked up by the lymph nodes and by the liver parenchyma, and we currently have in progress studies to attempt to determine just what dosage of colloidal gold and iodinated albumin will be most effective in eradicating experimental carcinoma and, in particular, how high the dosage can be raised without deleterious effect locally and on the liver and other vital structures.

IDENTIFICATION OF REGIONAL LYMPH NODES IN THE TREATMENT OF BRONCHIOGENIC CARCINOMA

JOSEPH A. WEINBERG, M.D.
LONG BEACH, CALIF.

CARCINOMA is essentially a local disease in its early manifestations. It tends to be confined to the region of its origin and to the lymphatics which receive lymph from the involved organ. Therefore, a prime consideration in the surgical treatment of cancer is the thorough removal of the regional lymphatics. The importance of this principle of treatment is recognized and practiced in most situations in which cancer occurs, but it has received litt'e attention in the surgical treatment of cancer of the lung. In a personal communication published in Taylor and Nathanson's monograph on "Lymph Node Metastases" in 1942, Churchill and Adams stated "the surgery of carcinoma of the lung, so far as the problem of metastases is concerned, is comparable to the treatment of carcinoma of the breast by simple mastectomy." The authors of the monograph go on to say, "technical advances will probab'y make the operation safer and may permit removal of most of the nodes likely to harbor metastases. Although the prospect is discouraging because of the inaccessibility of the node metastases, and the frequency with which they occur, these are the avenues which are open to progress."[1] Although it is almost a decade since these statements appeared, little or no progress has been made in this direction. I have been unable to find in the literature more than casual remarks to the effect that the regional nodes should be removed in pulmonary resection for bronchiogenic cancer, except the excellent description of the nodes given by Tuttle and Womack in 1934,[2] and the reference to the removal of specific groups of nodes contained in Jones' article published in 1947.[3]

During the past two and one-half years I have been studying the arrangement of the regional lymphatics of the lung by means of vital staining during surgery.[4] This study, augmented by studies of the pattern of lymph node metastases in cases of advanced bronchiogenic cancer, and reviews of the detailed descriptions of the lymphatics of the thorax made by Rouviere[5] and Delamere, Poirier, and Cuneo,[6] has resulted in the establishment of a technique which permits the exposure and removal of the several groups of lymph nodes on each side of the thorax which are most likely to be affected by metastases.

From the Veterans Administration Hospital, Long Beach, Calif., and the University of California at Los Angeles Department of Surgery.

This study was supported in part by a grant from Wyeth Incorporated, 1600 Arch Street, Philadelphia 3, Pa.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read at the Thirty-first Annual Meeting of The American Association for Thoracic Surgery, Atlantic City, New Jersey, April 16-18, 1951.

The procedure of vital staining of the thoracic lymphatics consists of the injection of pontamine sky blue into the lung as soon as the hemithorax is opened and the diagnosis is established. The dye is seen in the regional nodes within a few minutes after the injection is made. In some instances the staining of the nodes is preceded by blue streaking along the lymphatic channels. The vital staining makes it possible to recognize the location of the various groups of nodes which receive lymph from the lung. While most of the thoracic nodes can be recognized by their anthracotic pigmentation, this is not always sufficient for easy identification because of the tendency of the dust particles to be concentrated in the more central portion of the nodes. This tendency, together with the obscuration by the unpigmented capsule of the nodes and the "camouflaging" resulting from scattered anthracotic pigmentation in the perinodal tissues, often make recognition of very small nodes or deeply placed nodes difficult or impossible. The dye does not stain carcinomatous tissue, and stained nodes which contain deposits of malignant cells reveal the uncolored cancerous deposits against the blue background.

Pontamine sky blue, which is used for the vital staining, has the advantage of being only slightly diffusible when it is injected into the tissues. It tends to remain in the area in which the injection is made unless it is picked up by the lymphatic circulation. Once it is deposited in a lymph node it tends to remain there. Hyaluronidase (Wydase*) is added to the dye solution to accelerate its uptake by the lymphatics. The dye has no toxic effects in more than one thousand times the quantity used clinically for vital staining as determined by animal experiments.[7]

The only deleterious effect we have seen is the occasional slight pigmentation of the skin which probably results from accidental injection of the dye into a blood vessel. In the several cases in which this occurred the skin assumed a cyanotic appearance which cleared up in several days to a few weeks. It has never been a troublesome complication.

The technique of the staining procedure is carried out in the following manner. Five cubic centimeters of a 4 per cent solution of pontamine sky blue in distilled water is mixed with thirty units of Wydase. Three to five cubic centimeters of the mixture is injected superficially and deeply in the circumference of the pulmonary tissue near the root of the lung. Some of the dye is also injected against and into the wall of the main bronchus. Nodes and lymphatic vessels are seen to be stained within one or two minutes after the injection is made, but the staining of the more distant nodes is usually not apparent until ten to twenty minutes have elapsed. Excursions of the lung induced by the anesthetist at this stage accelerate the movement of the dye toward the lymph nodes. It is well to wait ten minutes or so after the injection before proceeding with the dissection in order to allow the dye to reach the more distant nodes. In the meantime one examines the mediastinum to determine the gross extent of cancer invasion.

*A product of Wyeth Incorporated, Philadelphia, Pa.

J. THORACIC SURG.
VOL. 22, NO. 5, NOVEMBER, 1951

A, The nodes in the upper two rows show vital staining with the blue dye. Very small stained nodes are seen in the top row, some of them against a background of unstained areolar tissue. In the lowest row, a mass of areolar tissue is seen on the left, and an unstained node is seen on the right, demonstrating the difficulty of differentiating the unstained node from areolar tissue.

B, The upper row shows small stained nodes against a background of areolar tissue.

The lower row shows two nodes on the left which are incompletely stained due to infiltration with cancer. The two stained nodes on the right are free from cancer.

C, Phrenic nerve with stained nodes along its course.

Fig. 1.

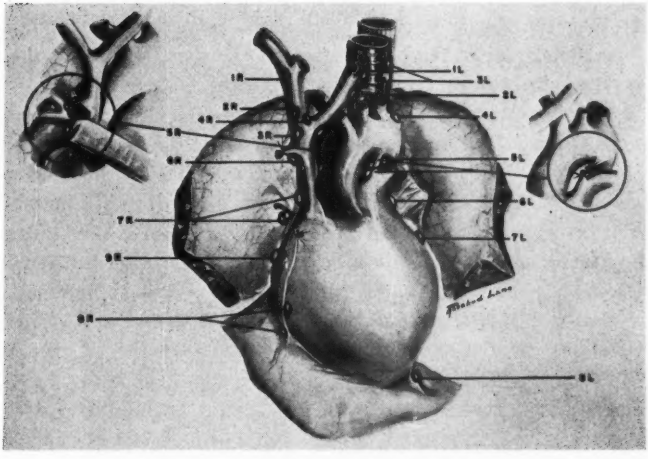


Fig. 2*A*.—Anterior view of regional lymph nodes of lungs and bronchi. *1R*-para-subclavian; *2R*—para-innominate; *3R*—paratracheal; *4R*—anterior mediastinal; *5R*—lower paratracheal; *6R*—azygos; *7R*—hilar; *8R*—nodes along phrenic nerve; *9R*—nodes of pulmonary ligament.

Left Side—*1L*—parasubclavian; *2L*—para-innominate; *3L*—paratracheal; *4L*—anterior mediastinal; *5L*—aortic window; *6L*—hilar; *7L*—nodes of pulmonary ligament; *8L*—nodes along phrenic nerve.

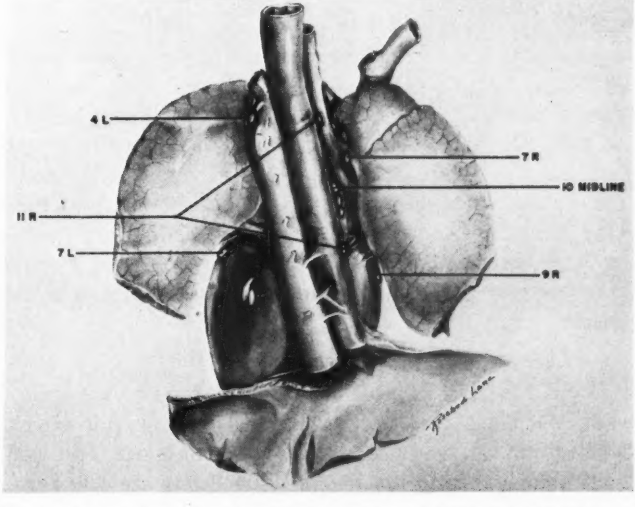


Fig. 2*B*.—Posterior view of regional lymph nodes of lungs and bronchi. *4L*—left anterior mediastinal; *7R*—right hilar; *7L*—nodes of left pulmonary ligament; *10*—midline, subcarinal; *11*—para-esophageal.

There has been no uniformity of success in staining the various groups of nodes. Sometimes an excellent injection of most of the groups of nodes is obtained; at other times only a few of the nodes take the stain. Many factors participate in determining the distribution of the dye. Probably the most important is the blockage of lymphatic pathways with carcinomatous tissue. Another is the presence of inflammatory changes which act as a barrier to the passage of the dye. Possibly a third is occasional departure from the usual injection technique. With improvement in the technique, successful staining of the nodes occurs with greater frequency. The procedure of vital staining has been used in seventeen cases of pneumonectomy, in five cases of lobectomy and in sixteen cases of simple exploration for malignancy, and in twenty cases of other types of surgical disease of the lung. At one time or another, all of the groups of regional nodes, which have been described by Ruviere[5] and by Delamere and his associates[6] in their complete monographs on the anatomy of the lymphatics, have been visualized. Vital staining of nodes has been seen in the opposite hemithorax in two cases in which death occurred within eleven days after surgery. This cross-staining occurred through the subcarinal and anterior mediastinal nodes. The nodes which have been stained also correspond closely to the pattern of the metastatic spread seen in individuals succumbing to bronchiogenic cancer.

Studies made on cadavers demonstrate that it is possible to remove most of the groups of regional nodes within the hemithorax with relative safety, and that in most instances it is possible to remove the nodes in continuity with the lung. We have altered our technique of pneumonectomy in several respects in order to accomplish this.

The approaches which permit the exposure of the several groups of nodes in each hemithorax will be described with the techniques for right and left radical pneumonectomy. Our preference is to attack the most peripheral groups first, and to carry the dissection toward the hilum without interruption. This procedure has the advantage of lessening the opportunity for embolic cancer cells. It also results in better exposure of the great vessels of the pedicle by first removing the lymph nodes and adventitia which obscure them. A lateral approach is made through the fifth interspace, as this gives a better exposure of all sides of the root of the lung than is obtainable with an anterior or posterior approach.

RIGHT RADICAL PNEUMONECTOMY

A longitudinal incision is made in the mediastinal pleura from the cupula to the azygos vein along the lateral margin of the superior vena cava. The nodes and adventitia on the under side of the subclavian vein (*1R*) and the nodes at the junction of the innominate veins (*2R*) are freed by sharp and gentle blunt dissection.

The dissection of the nodes and adventitia is continued downward en bloc to include the paratracheal (*3R*) para-esophageal (*3R*) and anterior mediastinal

nodes (*4R*). Most of this dissection is facilitated by retracting the superior vena cava to the left.

When the azygos vein is reached, this vessel is divided between ligatures close to the superior vena cava, and the vena cava is retracted to the left to expose the important group of paratracheal nodes (*5R*) which lie in the space between the superior vena cava anteriorly, the trachea and the right pulmonary

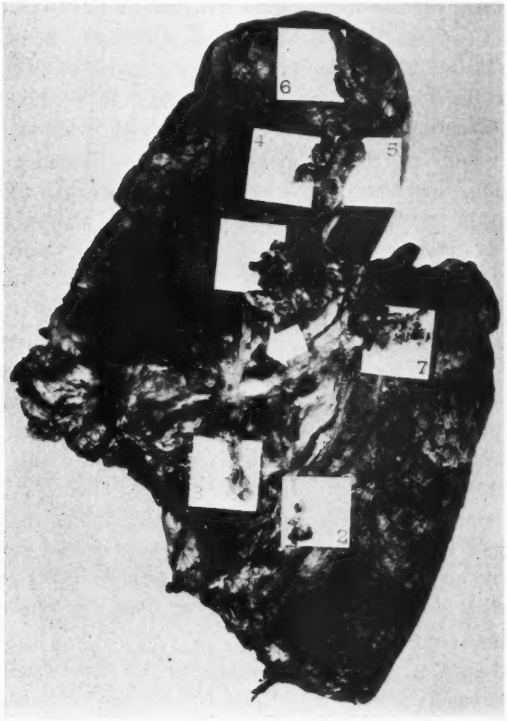


Fig. 3.—Right radical pneumonectomy with inclusion of regional lymph nodes. Arrow points to primary lesion in middle lobe bronchus. *1*—hilar nodes; *2*—nodes of pulmonary ligament and paraesophageal nodes; *3*—nodes along phrenic nerve; *4*—azygos nodes; *5*—paratracheal and paraesophageal; *6*—nodes along right subclavian and innominate veins; *7*—subcarinal nodes.

artery posteriorly, and the aortic arch to the left. The block dissection is continued downward toward the hilum to include this group and, at the same time, the nodes around the termination of the azygos vein (*6R*).

The right pulmonary artery which has been well exposed as a result of the preceding dissection is now doubly ligated proximally and distally, and is divided proximal to its branching. After dividing the mediastinal pleura over the anterior part of the pulmonary root, the hilar nodes (*7R*) and adventitia are thoroughly removed in this region and are left in continuity with the lung.

At this stage of the operation the exposed main bronchus is clamped to prevent the passage of secretions into the contralateral lung.

The phrenic nerve and the nodes along its course (*8R*) are removed with the vessels and areolar tissue accompanying the nerve. Although the direction of lymph flow is from the diaphragm toward the tracheobronchial nodes, the lymph vessels accompanying the nerve frequently show staining with the vital dye, and metastases have been seen in nodes along the phrenic nerve on several occasions. For these reasons it is advisable to remove these structures routinely.

The anterior and posterior leaves of the pulmonary ligament are divided close to the mediastinum in order to remove the nodes of the pulmonary ligament (*9R*) and the local paraesophageal nodes. In accomplishing this, the nodes and adventitia are maintained in continuity with the lung.

The inferior and superior pulmonary veins are ligated and divided close to the pericardium, care being exercised to dissect away the hilar nodes in this region and maintain their continuity with the lung. If the pulmonary veins are short and there is difficulty in dividing them, it is better to divide them within the pericardial sac rather than through their tributaries; otherwise one will have difficulty in including all of the hilar nodes with the resected specimen.

The lung is now retracted lateralward and upward in order to expose and remove the subcarinal nodes (10 midline). It is usually possible to remove all of these nodes well onto the under side of the left main bronchus. The nodes on the posterior aspect of the root of the lung are exposed by removing the mediastinal pleura from the posterior surface of the pedicle if this has not already been done. This will also expose the paraesophageal nodes (*11R*) lying above and below the level of the main bronchus. The esophageal nodes are not as likely to be involved as others, but, having seen them take up the dye, and having seen them involved with malignancy, we make it a practice to remove them together with the nodes on the posterior aspect of the pulmonary root.

The bronchus is now divided close to the trachea, and the stump is closed with interrupted sutures. Careful inspection is made for bleeding, particularly for bleeding from small vessels which have been interrupted during the removal of groups of lymph nodes. After it is ascertained that the field is dry, the intercostal incision is closed without drainage.

LEFT RADICAL PNEUMONECTOMY

The general plan of resection is the same as for right radical pneumonectomy, the differences being that there is a very important group of nodes within the "aortic window" to deal with on the left side, and there is no vessel which corresponds to the superior vena cava.

The mediastinal pleura is incised from the cupula to the upper limit of the pulmonary root. The left subclavian (*1L*) and innominate paravenous nodes (*2L*) are excised in the manner which has been described for the right side, and the dissection is continued toward the hilum, including the para-

tracheal (*3L*) and anterior mediastinal nodes (*4L*). A node is almost always found on the dome of the arch of the aorta near the origin of the left subclavian artery.

The nodes in the "aortic window," the space within the arch of the aorta bounded inferiorly by the left pulmonary artery, are thoroughly removed (*5L*). This group of nodes commonly receives metastases, particularly if the left upper lobe bronchus or main bronchus is the source of the malignancy. This dissection is performed by first dividing the ligamentum arteriosum between ligatures in order to obtain a free approach to the depth of the space. The nodes are removed together with those over the left pulmonary artery and bronchus. These

Fig. 4.—Left radical pneumonectomy with inclusion of regional lymph nodes. Primary lesion in apex of upper lobe. *1*—hilar nodes; *2*—nodes within aortic window; *3*—subcarinal nodes; *4*—paratracheal and anterior mediastinal nodes; *5*—nodes of pulmonary ligament and paraesophageal nodes. The phrenic nerve which lies to the right of the main bronchus is not labelled.

nodes can usually be kept in continuity with the lung. One should carry the dissection within the arch of the aorta only to a distance which is considered safe for the control of bleeding from small vessels which may be interrupted. There is no apparent dividing line between these nodes and the right paratracheal nodes, and one must estimate the limit to which the dissection should be carried. There is probably no advantage in including right paratracheal

nodes, since there is no natural communication between them and the nodes of the left side. After completing the removal of the nodes in the "aortic window," the exposed left pulmonary artery can be seen to its origin from the main pulmonary artery.

The remainder of the resection is carried out in the same manner as on the right side.

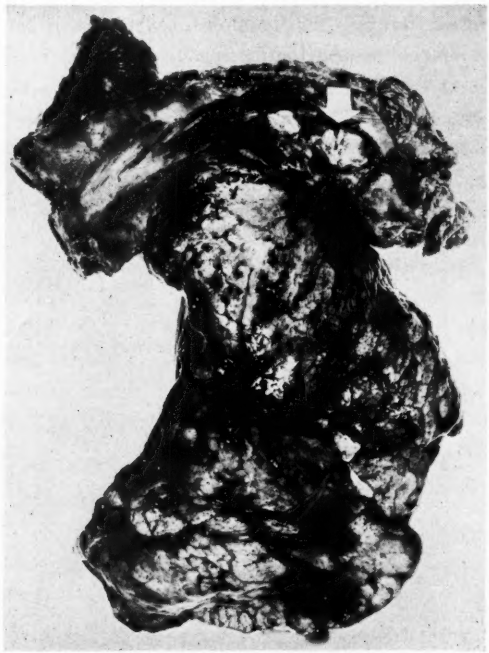


Fig. 5.—Cancer adherent to fourth rib, laterally. The third, fourth, and fifth ribs with parietal pleura, anterior and posterior intercostal nodes, and muscles overlying resected ribs have been removed en bloc with the resected lung and regional lymphatics.

RADICAL PNEUMONECTOMY WITH INCLUSION OF SEGMENT OF THORACIC WALL

The only nodes which remain to be considered are those of the parietal wall. While the parasternal nodes have taken up the dye in some instances, this has not been a frequent concurrence. The direction of the flow of lymph from the lung is not toward the nodes of the parietal wall of the thorax. We have limited the excision of the anterior and posterior chains of intercostal nodes to cases in which the carcinoma has become attached to the parietal pleura. When this situation is encountered, both chains are removed with a portion of the thoracic wall which is left attached to the lung. The excision is made en bloc, including at least one rib above and one rib below the site of attachment of the neoplasm. The parietal pleura, the muscles overlying the re-

sected ribs, and the intercostal nodes in the segment of thoracic wall which is involved, are excised. There remains only subcutaneous tissue and skin over the area at the completion of the procedure.

While we have not yet performed dissections in the neck to remove the jugulo-subclavian nodes, this step is a logical one if nodes in the chain of communication from the lungs are to be removed as thoroughly as it is possible to do so. This might be performed several days before pneumonectomy.

There has been one fatality which can be ascribed to the extensiveness of the nodal dissection. This was in a patient with advanced cancer with metastatic involvement of most of the mediastinal nodes, in which all visible nodes were removed from under the superior vena cava. The patient died sixteen hours after operation, and at autopsy the hemithorax was found to be filled with clotted blood. No open vessels were discovered. It is assumed that bleeding from small vessels in the mediastinum caused the patient's death. Since this occurrence we routinely place pledgets of "gelfoam" soaked in thromboplastin in the space underlying the superior vena cava after removing the nodes from this area.

We have no statistics on longevity to report in these recent operations with vital staining as an adjunctive procedure. The emphasis which we have placed on extensive regional node resection is based solely on the premise that cancer, particularly cancer of the lung, spreads essentially by way of the lymphatics. It will take many years of experience with the procedure to permit a significant comparison of results of pneumonectomy with and without the inclusion of the several echelons of nodes which receive lymph from the lung.

SUMMARY

A method is described which is designed to facilitate the identification of regional lymph nodes of the lung for their removal during the operation of radical pneumonectomy for bronchiogenic carcinoma.

A solution of pontamine sky blue dye and hyaluronidase is injected into the pulmonary parenchyma as soon as the thorax is opened. The dye is taken up by the regional lymph nodes, imparting to them a blue color which makes them more easily distinguishable than they would be in their natural state. Many nodes which would remain obscured, even with acquired dust pigmentation, become readily identifiable after being colored by the dye.

The mapping of the nodes by vital staining, augmented by studies of the pattern of lymph node metastases in cases of advanced bronchiogenic cancer, has resulted in the establishment of a technique which permits the exposure and removal of the several groups of lymph nodes on each side of the thorax which are most likely to be affected by metastases.

This method is offered as a means of more nearly approaching the ideal of planned resection of lymph nodes, a phase of the operation which is of fundamental importance in the surgical treatment of bronchiogenic carcinoma.

REFERENCES

1. Taylor, C. W., and Nathanson, J. T.: Lymph Node Metastases, New York, 1942, Oxford University Press.
2. Tuttle, Wm., and Womack, W. A.: Bronchiogenic Carcinoma, J. THORACIC SURG. 4: 125, 1934.
3. Jones, J. C.: Surgical Aspects of Bronchiogenic Carcinoma, J. A. M. A. **134**: 113, 1947.
4. Weinberg, J., and Greaney, E. M.: Identification of Regional Lymph Nodes by Means of a Vital Staining Dye During Surgery of Gastric Cancer, Surg., Gynec. & Obst. **90**: 561-567, 1950.
5. Rouviere, H.: Anatomy of the Human Lymphatic System, Ann Arbor, 1938, Edwards Bros.
6. Delamere, C., Poirier, P., and Cuneo, B.: The Lymphatics, London, 1913, Constable and Co.
7. Weinberg, J., Greaney, E. M., Rawlings, B., and Haley, T. J.: The Use and Toxicity of Pontamine Sky Blue. (To be published in Science.)

TEMPORARY UNILATERAL OCCLUSION OF THE PULMONARY ARTERY

A New Method of Determining Separate Lung Function and of Radiologic Examinations

Eric Carlens, M.D., Hans Erik Hanson, M.D., and Björn Nordenström, M.D.
Stockholm, Sweden

WHEN elective surgery, which will impair the function of a lung is being considered, the functional state of the contralateral lung should always be taken into account. A simple estimate is often enough, particularly when the patient is young and the remaining lung exhibits no symptoms of disease. On the other hand, if the function of the remaining lung is doubtful, the examination should be rather more thorough, especially in cases where a certain amount of cardiorespiratory insufficiency already is present before the operation. In addition to the ordinary lung tests, it may sometimes be indicated to make a complete study of the circulation including cardiac catheterization.

However, no accurate estimate of the function of either lung can be obtained save by bronchospirometry. The latter method is, in its present state of development, capable of providing fairly accurate figures for the vital capacity of each lung and for its percentage participation in the absorption of oxygen. The bronchospirometric values recorded in air inhalation sometimes deviate slightly from those in oxygen inhalation. The former values are surely the most physiologic ones, but the difference may yield some information regarding the ratio between the volumes of residual air in the two lungs.

If bronchospirometry were combined with work, it seems likely that the maximal oxygen-absorbing power could be determined for each lung separately. Yet in the experiments so far carried out during moderate work (oxygen absorption four to five times normal) the percentage participation of each lung in the absorption of oxygen did not differ from conditions at rest (Björkman and Carlens[1]).

However, even after a study of the circulation, and having recourse to all conceivable information about the function of each lung, it will always be a matter of opinion whether or not a diseased lung can be removed in the individual case. In doubtful cases it would accordingly be highly desirable if it were possible before the operation temporarily to put the patient in a state which so far as possible resembled the functional condition he would be likely to be in after the operation. Attempts have been made to achieve this result by occluding the main bronchus to the diseased lung, or by letting it inhale

From the Department of Surgery (Head, C. Crafoord, M.D.), the Department of Oto-Laryngology (Head, G. Öhngren, M.D.) and the Department of Radiology (Head, B. Holmgren, M.D.) of Sabbatsberg Hospital, Stockholm, Sweden.
Received for publication April 24, 1951.

nitrogen and the healthy lung oxygen while the bronchospirometric study is in progress (Jacobaeus and Bruce[2]). To prevent oxygen excretion from the lung which is breathing pure nitrogen, it has proved preferable to substitute artificial alveolar air for the nitrogen. The former gas mixture and the venous blood are in balance with one another, and no absorption or excretion can take place from the diseased lung. If the healthy lung thereby can be made to take care of the entire respiratory work without troubling the patient unduly, the test is a good one. Although these experiments can be made at rest, it

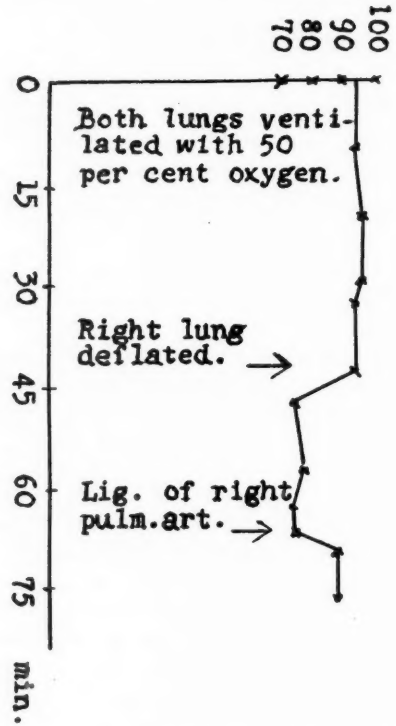


Fig. 1.—Oxygen saturation of the arterial blood during a right-sided pneumonectomy with pentothal-N_2O-curare anesthesia. Ventilation is maintained by spiropulsator through a double lumen tube to each lung separately.

is probably impossible to determine the respiratory reserve by carrying them out during physical work owing to the severe strain imposed on the patient. If the diseased lung functions comparatively well and its supply of oxygen is cut off under maintained blood circulation, the oxygen saturation of the mixed arterial blood will be reduced. Such admixture of venous blood from the occluded lung is most pronounced at first, but animal experiments have demonstrated that it is almost wholly compensated after about eight hours, since the circulation ceases almost completely in the nonrespiring lung (Euler and

Liljestrand,[3] Dirken and Heemstra,[4] Nisell[5]). However, there is much evidence to show that this reflex mechanism does not always operate in the same perfect manner in an abnormal lung in a middle-aged or elderly patient. Fig. 1 shows the arterial oxygen saturation as recorded by an oximeter, in a patient 67 years of age who was undergoing pneumonectomy under pentothal-nitrous oxide anesthesia. The patient's respiration was blocked by curare and maintained by a spiropulsator. A double-lumen tube enabled the lungs to be ventilated separately (Björk and Carlens[6]). Both lungs were ventilated at first, but as soon as the thoracic wall was opened up the healthy lung only was ventilated. At the very same moment the arterial oxygen saturation began to decline, although the inhaled gas consisted of 50 per cent oxygen; and it did not again begin to rise until the shunt had been bypassed by ligation of the pulmonary artery on the operated lung (Carlens and Finnerty[7]).

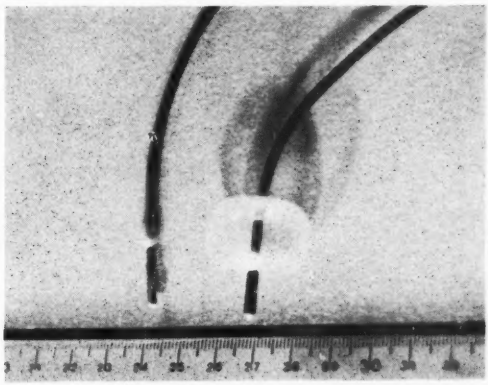


Fig. 2.—Photograph of cardiac catheter provided with cuff (inflated).

In some such cases, therefore, it seems likely that the patient will do well after extirpation of the diseased lung, though he cannot tolerate the effect when its oxygen supply is discontinued preoperatively. This behavior is due to the presence of a venous shunt in the latter case. Thus, theoretically, a stoppage of the function of the diseased lung by occlusion of its branch of the pulmonary artery would seem to be less trying to the patient and more in line with postoperative conditions when all the venous blood flows to the remaining, respiring lung. So far as we can find no such experiments have hitherto been made but, technically, the procedure should offer no serious difficulties if the method of heart catheterization is adopted.

METHODS

The opening in the tip of an ordinary Cournand cardiac catheter was filled in and a few centimeters proximally of it another one was made. Over it was placed an 8 mm. long, inflatable cuff of strong and thin latex rubber. When inflated the cuff assumed the shape of a short cylinder having a diameter of up to 3 cm.

The catheter was inserted in the usual manner in an exposed elbow vein or, in the preliminary experiments on dogs, in an exposed vein in the groin. Under fluoroscopic control the catheter could at will be made to enter the right or the left branch of the pulmonary artery. The rubber cuff was then slowly distended by injection of a 35 per cent solution of Umbradil through the catheter. This makes it easy to follow the distention and shape of the rubber balloon on the fluoroscopic screen. Consequently, the risk of excess pressure in the cuff is small and therefore damage to the vascular wall need not be feared.

The experiments on dogs were performed under barbiturate anesthesia, and 1 ml. of heparin was given before the catheterization to prevent clotting of the stagant blood distal to the balloon.

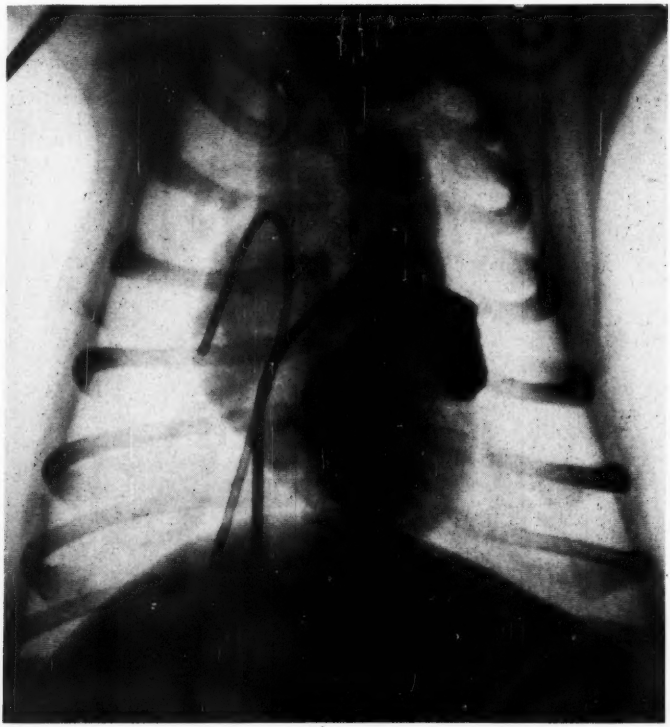


Fig. 3.—Roentgenogram of a dog with an inflated balloon in the left pulmonary artery and a conventional catheter in the right one. Both catheters inserted through the saphenous vein.

RESULTS

Ten experiments were performed on eight dogs, and in no case could we observe any complications following unilateral occlusion of the pulmonary artery. In one case one of the main branches was kept in a state of total occlusion for two hours, and in another it was impossible to find any electrocardiographic changes or other signs of complication despite very rapid and hard inflations.

How the pressure varies in the pulmonary artery has not yet been studied in detail, but in a few cases another catheter was introduced into the main trunk of the vessel, a slight increase in pressure being registered in immediate association with occlusion of one of the main branches.

In some instances the study was supplemented with cardioangiography. The contrast medium was injected into an exposed vein in the contralateral

Fig. 4.—An angiocardiogram of a dog with inflated balloon in the right pulmonary artery. The catheter is inserted in the saphenous vein on one side and on the other side is a contrast medium (Umbradil) injected and has filled the pulmonary vessels on the left side only.

groin, and, as revealed by Fig. 4, the contralateral branches of the pulmonary artery were filled selectively. This is at least a rough indication that no blood passed beside the inflated cuff.

After a slight modification of the bronchospirometry catheter for use in human beings (Carlens[8]), we were able to perform bronchospirometry on two dogs in conjunction with unilateral occlusion of the pulmonary artery.

Fig. 5 shows such a registration when the right pulmonary artery was occluded by the inflatable cuff on the cardiac catheter. The ventilation was somewhat greater on the right side, but oxygen absorption had been taken over by the left lung. The right respirometric tracing was perfectly horizontal. As soon as the occluding balloon had been emptied by suction, the right respirom-

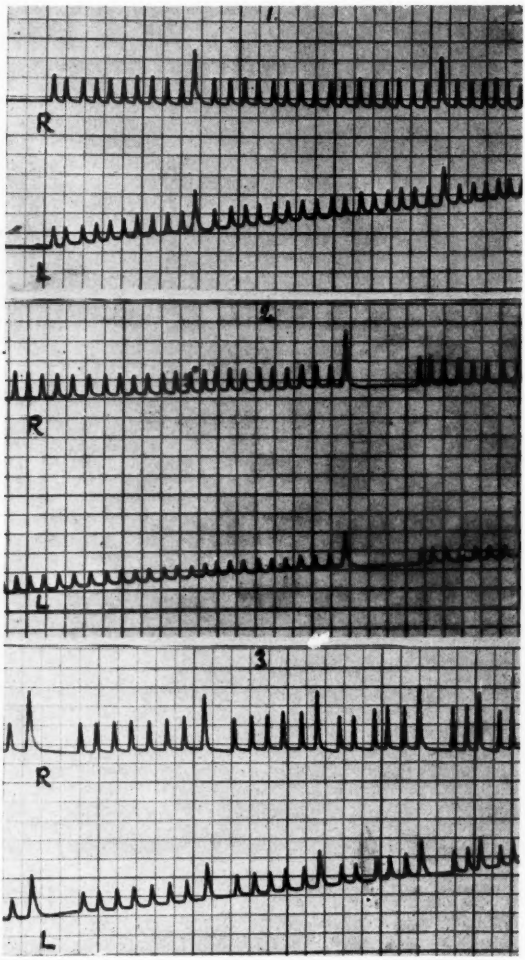


Fig. 5.—Bronchospirometric tracings in a dog with a cardiac catheter with inflatable cuff in the right pulmonary artery. *1*, Cuff inflated. No oxygen uptake in the right lung. *2*, Cuff deflated. Oxygen uptake in both lungs. *3*, Cuff reinflated. Oxygen uptake in left lung only.

etric curve rose as a sign of resumed oxygen absorption from the ipsilateral lung, and it at once declined when the balloon was reinflated. The total amount of oxygen absorbed per unit time remained constant whether or not the right branch of the pulmonary artery was occluded, demonstrating that

the nonoccluded lung immediately took over the entire function. Such a bronchospirometric study should be a good check on the completeness of the fluoroscopically visualized occlusion.

No complications could, as noted, be observed, and all the dogs survived the operation without any visible disability. On two dogs the experiment was repeated with the same results after two weeks. One dog, killed three days after

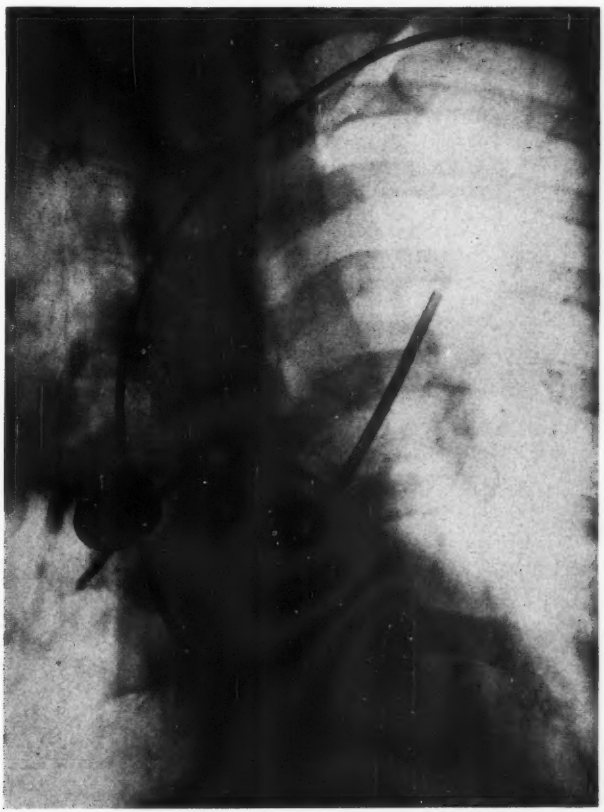


Fig. 6.—A catheter with inflated balloon in the right pulmonary artery in a patient who has had thoracoplasty on the right side. In addition a conventional catheter in the left pulmonary artery inserted through the saphenous vein.

the experiment, was autopsied, but no macroscopical changes were observed in the occluded lung. Nor were any changes discovered by microscopical inspection of the vascular wall at the site of the inflated balloon.

EXPERIMENTS ON MAN

Since our animal experiments in no case exhibited any signs of complications, we considered it justified under the strictest precautions to try the method on three patients, who were considered for pneumonectomy.

Catheterization and occlusion of a main branch of the pulmonary artery were performed under local anesthesia after premedication with nembutal. The patients were given 3 ml. of a 5 per cent solution of heparin intravenously. The pulmonary artery was occluded for periods lasting up to twenty minutes. None of the patients experienced any subjective embarrassment from the chest. The rate and character of the pulse remained unchanged in occlusion, and so did the blood pressure in the brachial artery. In one case the mean pressure in the pulmonary artery, measured through a second catheter, was seen to rise from 17 to 22 cm. of water, but it regained its initial level after ten

Fig. 7.—A roentgenogram of a patient with a catheter with inflated balloon in the left pulmonary artery. In addition contrast medium (Umbradil) has been injected through a conventional catheter in the superior vena cava and has filled the vessels on the right side only.

minutes. In one case no rise in pressure was recorded when the pulmonary artery was subjected to unilateral occlusion. In one case electrocardiogram tracings were recorded in three standard leads while the pulmonary artery was occluded repeatedly. There were no electrocardiogram alterations. In no case were there any complications following the experiment. None of the patients suffered any distress other than the sensations that usually accompany cardiac catheterization.

Thus, the preliminary results seem to indicate that it is possible in conscious human beings to occlude both the circulation in the pulmonary artery

and the uptake of oxygen in one lung, and thereby approach as close as possible to the condition following a contemplated pneumonectomy. Under such conditions the circulation in the bronchial artery is not occluded, and consequently there is no need to fear lesions due to anoxia in the pulmonary tissues.

We intend, therefore, by this method to study the degree of dyspnea, the oxygen saturation of the arterial blood, the pressures, and resistances in the greater and lesser circulations at rest and during exercise.

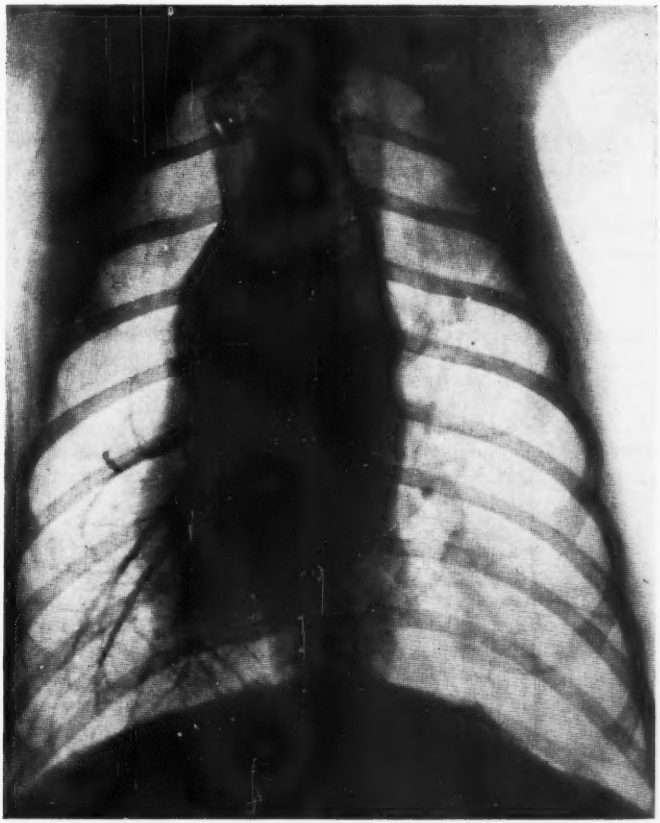


Fig. 8.—An ordinary roentgenogram of a dog with a double lumen catheter in the left pulmonary artery. The balloon is inflated and a small amount of a weak contrast solution is injected peripherally to it.

It is possible, moreover, that unilateral occlusion of the pulmonary artery may have radiological applications. By the methods used currently it may be impossible to visualize the pulmonary artery on one side in a lateral view, since the right and left sides are superimposed. By injection proximal to the occluding balloon the pulmonary vessels on one side can be filled with contrast medium. Contrast material may also be injected in the occluded pulmonary

artery distal to the balloon, whereby it is unnecessary to use large amounts of highly concentrated contrast solution rather than small quantities of a solution which is isotonic with the tissues. There is no need of rapid serial exposures. The method is therefore likely to open up a means of studying in detail the vascular bed in the lungs. Experiments along these lines are at present in progress.

SUMMARY

In a preliminary experimental investigation on dogs, and in three cases on human beings, the pulmonary artery was occluded on one side by means of a cardiac catheter provided with an inflatable balloon. By this means the circulation in the pulmonary artery on one side and the oxygen uptake in the ipsilateral lung could be stopped. Thereby conditions were created which closely resembled the status following pneumonectomy, so that circulatory and respiratory changes resulting from extirpation of one lung could be foretold. No complications were observed.

It is possible that unilateral occlusion of the pulmonary artery may constitute a means for unilateral contrast radiography of the pulmonary vessels.

ADDENDUM

Since writing this paper, an article has been published by Charles Dotter and Daniel Lucas in Am. J. Physiol. **164:** 254, 1951. These authors have used a similar technique and have done an experimental investigation on dogs. After occlusion of either the right or the left pulmonary artery they registered in every case a hypertension in the right ventricle and they did not continue the experiments more than three minutes because of grave symptoms of cor pulmonale in the dogs. Judging from the published photograph and figures, we feel certain that there must have been a total or subtotal occlusion of the main stem of the pulmonary artery in every case.

REFERENCES

1. Björkman, S., and Carlens, E.: The Lung Function During Rest and Exercise in Lung Diseases. A Bronchospirometric Study, Acta Med. Scandinav. Suppl. **259:** 59, 1951.
2. Jacobaeus, H. C., and Bruce, T.: A Bronchospirometric Study on the Ability of the Human Lungs to Substitute for One Another, Acta Med. Scandinav. **105:** 193, 1940.
3. Euler, U. S., v., and Liljestrand, G.: Observations on the Pulmonary Arterial Blood Pressure in the Cat, Acta Physiol. Scandinav. **12:** 301, 1946.
4. Dirken, M. N. J., and Heemstra, H.: Alveolar Oxygen Tension and Lung Circulation, Quart. J. Exper. Physiol. Cogn. Med. Science **34:** 193, 1948.
5. Nisell, O.: The Action of Oxygen and Carbon Dioxide on the Bronchioles and Vessels of the Isolated Perfused Lungs, Acta Physiol. Scandinav. **21:** Suppl. 73, 1950.
6. Björk, V. O., and Carlens, E.: The Prevention of Spread During Pulmonary Resection by the Use of a Double-Lumen Catheter, J. THORACIC SURG. **20:** 151, 1950.
7. Carlens, E., and Finnerty, J.: In press.
8. Carlens, E.: A New Flexible Double-Lumen Catheter for Bronchospirometry, J. THORACIC SURG. **18:** 742, 1949.

OBSERVATIONS ON THE HEMODYNAMICS OF BRONCHIAL-PULMONARY VASCULAR COMMUNICATIONS

DONALD P. SHEDD, M.D.,* RALPH D. ALLEY, M.D.,**
AND GUSTAF E. LINDSKOG, M.D.
NEW HAVEN, CONN.

IN THE course of certain experimental preparations in the dog intended to elucidate problems in pulmonary congestion, with particular reference to its effect on collateral respiration, rather unexpected and apparently paradoxical findings were encountered with regard to pressure changes in the pulmonary vascular tree. In brief, after complete occlusion of a lobar pulmonary artery and its corresponding veins, a progressive and striking elevation of endarterial pressure occurred in the occluded portion of the pulmonary vascular bed. This phenomenon seemed to us best explainable by the existence of a very active and immediate collateral circulation to the pulmonary vascular bed from the bronchial arteries at high systemic pressures. It is the purpose of this paper to describe further experiments employed in studying this mechanism, to present in detail the technical methods employed, to set forth the experimental data obtained, and to discuss the significance of these findings in relation to normal pulmonary hemodynamics.

Microscopic vascular communications between the bronchial and pulmonary arterial systems at the capillary level have long since been described in the normal lung.[1-4] After interruption of the pulmonary artery[5] and in various pathologic states these, and possibly newly developed channels, reach macroscopic proportions.[6, 7] All previous observations on such anastomoses were obtained by study of microscopic serial sections, or by injection and perfusion techniques[4, 8] The literature to our knowledge contains no reports of in vivo experiments designed to record endovascular pressure changes within an otherwise normal pulmonary lobe after occlusion of both arterial inflow and venous outflow.

MATERIALS AND METHODS

All experiments were performed on presumably healthy stock dogs weighing 13 to 28 kg. under sodium barbital anesthesia (300 mg. per kilogram), employing intravenous sodium pentothal for induction. Thoracotomy was done by resection of the left fifth or sixth rib under positive pressure respiration, which was discontinued at the time of closure of the wound. The technique for occlu-

Presented at the 83rd meeting of the Society of Clinical Surgery, New Haven, Conn., Nov. 11, 1950.
Aided by Office of Naval Research N6ori-44 Task Order XI.
Received for publication April 24, 1951.
*William Harvey Cushing Memorial Fellow—Surgery.
**James Hudson Brown Memorial Fellow—Surgery.

sion of the appropriate vessels and the manner of their catheterization underwent considerable evolution during the earlier experiments. These will be summarized in the next paragraph. The arrangement finally evolved for occlusion of the left lower lobe pulmonary artery and vein through the closed chest by a snare cannula (Fig. 1) about an indwelling pressure-recording catheter is depicted for the artery in Fig. 2, and for the vein in Fig. 3. The left lower lobe was selected for this technique, because its supplying pulmonary artery and vein are easily accessible, and the exposure not complicated by the presence of an

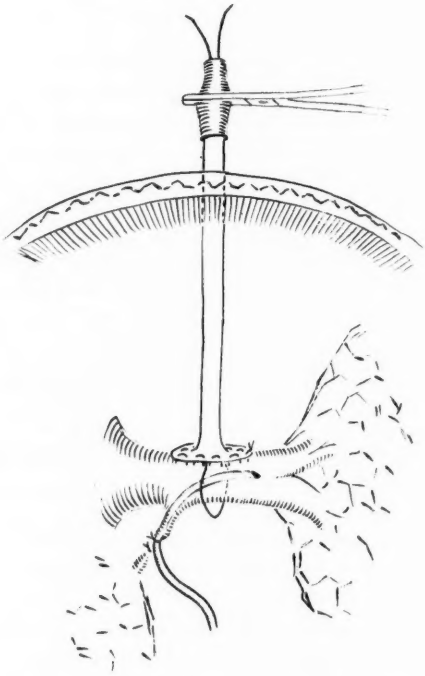


Fig. 1.—Diagram showing the polyethylene snare cannula sutured to the left lower lobe vein, with the snare loop passing through the cannula and around the vein. The end of the cannula is brought out through the thoracotomy wound with a tight closure of all layers around the cannula. To prevent ingress of air through the cannula, a rubber cuff surrounds its open end, which thus can be kept closed with a clamp except when manipulating the snare loop. The catheter for conducting intralobar pressures to the recording system is shown passing into the lower lobe via the right accessory lobe vein.

accessory or subcardiac lobe, as is the case on the right. Furthermore, advantage could be taken of the anatomic fact that the right accessory lobe vein usually joins with the left lower lobe vein close to the auricle. This permitted the retrograde introduction of a venous pressure-recording cannula through the sacrificed accessory lobe vein into the left lower lobe vein. The pulmonary artery pressures were measured in four experiments with a water manometer, and in nine subsequent experiments with a Hathaway multi-channel electronic

recorder.* Pulmonary vein pressures were recorded by the latter instrument in three experiments. Systemic blood pressure from a cannulated femoral artery was recorded by means of an ink-writing mercury manometer or with the electronic instrument. Intrapleural pressures were obtained by insertion through the left thoracic wall of an anchor-shaped glass cannula which was connected with an outlet of the electronic recorder. Continuous spirograms were recorded in three experiments employing an oxygen-filled Roth-Benedict spirometer with a one-way valve circuit and a CO_2 scrubber interposed connected to a tracheal cannula.

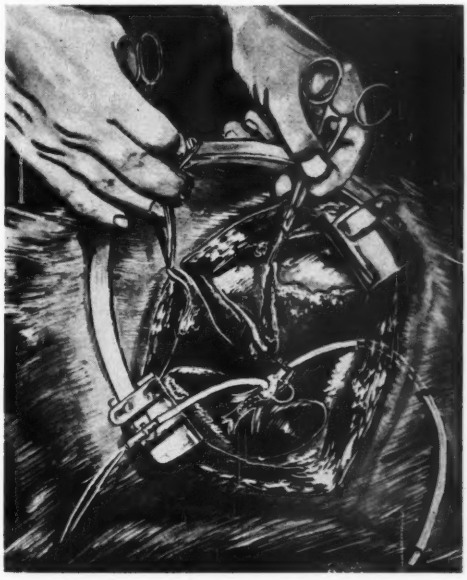


Fig. 2.—Showing the snare loop sutured to the left lower lobe artery, and the catheter passed via an upper lobe artery down into the lower lobe, past the point where the artery will be later occluded by pulling on the snare.

The principal variations in technique tested and subsequently discarded during the earlier experiments involved the manner of occluding the left lower lobe vessels. In the first experiment a double-lumen Cournand catheter with an inflatable balloon near its tip was introduced via the right external jugular vein during the thoracotomy, manually guided through the heart into the left lower lobe pulmonary artery, and the artery occluded by inflating the balloon. Because of the small lumen size of this catheter, an improvised double-lumen balloon-tipped catheter of polyethylene was employed in two subsequent experiments (No. 21 and 23). In the latter of these experiments a single lumen polyethylene catheter was also passed via the left auricular appendage into the left lower lobe pulmonary vein for recording its pressure, but it was found that dogs

*Hathaway Instrument Company, Denver, Colo.

tolerate intracardiac catheters poorly for long periods of time. Therefore, starting with experiment 24, cardiac catheters were discarded. Next, after an intercostal thoracotomy approach, short balloon-tipped polyethylene catheters were introduced through a left upper lobe artery, threaded down into the left lower lobe artery, and the other end of the catheter was brought out through a stab wound in the chest wall. Sizes of catheters were chosen such that the outside diameter of the catheter would be about one-third the inside diameter of the vessel, and the inside diameter of the catheter would admit a 15- to 17-gauge needle. The tiny homemade latex balloons proved troublesome, however, there being a tendency for the balloon to extrude itself proximally when inflated.

Fig. 3.—Showing the snare loop sutured to the left lower lobe vein at the hilus of the lobe, and the vein catheter passing via the right accessory lobe vein into the left lower lobe, beyond the point of later occlusion of the vein.

Therefore, the snare-cannula technique mentioned above was finally adopted for the lobar artery as well as the vein. This technique proved satisfactory, and was used in all subsequent experiments.

Two experiments (39 and 40) were chronic rather than acute. In these, simple ligation of the left main pulmonary artery was carried out five and six weeks, respectively, prior to the acute final pressure determinations, in order to study changes accompanying collateral expansion of the bronchial vascular system. At the time of the second thoracotomy in both animals, large, dilated, tortuous bronchial arteries were visible coursing over the hilar structures, mediastinum, and in the pulmonary ligament. The enlarged collateral vessels in the pulmonary ligament were sacrificed in the dissection exposing the right accessory lobe vein.

The effects of intravenous epinephrine, exsanguination, and subsequent intra-arterial retransfusion on intralobar pulmonary vascular pressures in the presence of occlusion of both lobar pulmonary artery and vein were also studied.

Following variable periods of hours during which the pressure studies were done, each animal was sacrificed and autopsied.

RESULTS

A. *Effect of Occlusion of Pulmonary Artery and Vein of the Left Lower Lobe in Normal Dogs.*—

Occlusion of a lobar vein alone caused an immediate moderate rise of intravascular pressure within the lobe, with a return to normal upon release of the vessel, as shown in Table I. The pressures reached exceeded the base line pressure by 3 to 18 mm. Hg.

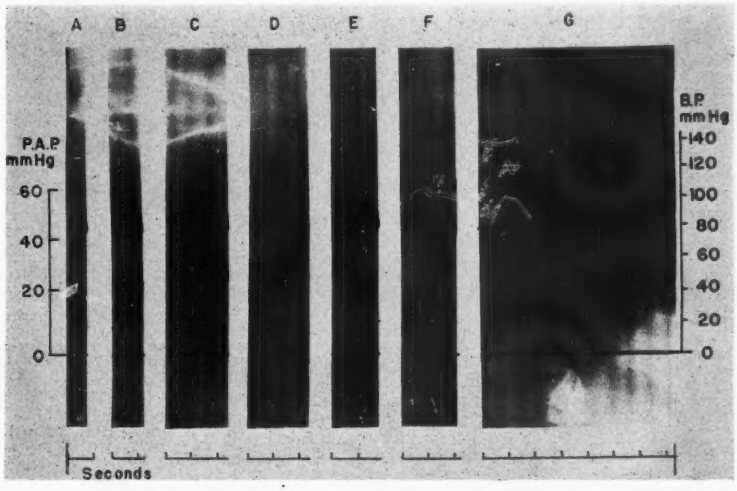


Fig. 4.—(Experiment 32.) Showing the effect of occlusion of the pulmonary vein and artery upon the pressure within the latter vessel distal to the point of occlusion. Upper tracing—systemic blood pressure (*B.P.*); lower tracing—pulmonary artery pressure within the obstructed area (*P.A.P.*). *A*, 12:33, base line. *B*, 12:35, after occlusion of pulmonary vein. *C*, 12:38, two minutes after occlusion of pulmonary artery, vein still occluded. *D*, 12:46, ten minutes after occlusion of pulmonary artery, vein still occluded. *E*, 12:47, eleven minutes after occlusion of pulmonary artery, vein still occluded. *F*, 12:51, fifteen minutes after occlusion of pulmonary artery, vein still occluded. *G*, 12:55, during release of pulmonary vein.

Occlusion of a lobar artery alone by intraluminal balloon or perivascular snare caused a prompt drop of pressure within the lobe, with disappearance of the pulse wave.

Occlusion of both lobar artery and vein, together in either sequence, was followed by a marked rise of pressure in the occluded segment to a level greatly exceeding the base line, pressures greater than 40 mm. Hg being reached in several instances as shown in Table I. A variable period of minutes was required for the maximum pressure to be reached (Figs. 4 and 5). From the Hathaway recorder tracings it was seen that at the height of pressure change a marked

respiratory wave was still present. In those experiments wherein observation was prolonged for several hours, the elevated pressures were maintained at the peak level. Upon release of the lobar vein obstruction, pressures promptly fell to subnormal levels, following which release of the lobar artery occlusion resulted in a return to normal levels (Fig. 4). This sequence of events could be repeatedly demonstrated on the same dog, and was consistent in all thirteen experiments but one. At autopsy in this animal it was noted that venous occlusion was incomplete because a vein draining the apical segment of the lower lobe had not been included in the snare loop.

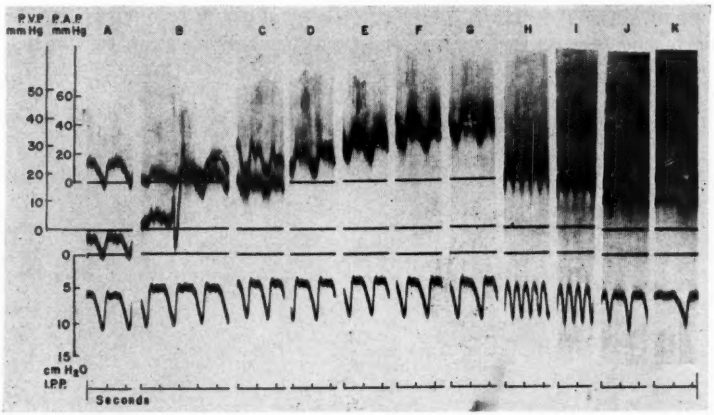


Fig. 5.—(Experiment 36.) Showing the rise in pulmonary artery and vein pressures produced by occlusion of both the artery and the vein. Also showing the effect upon the high pressure so produced within the lobe of systemic hypotension caused by bleeding. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural pressure (*I.P.P.*). *A*, 3:15, base line. *B*, 3:18, during occlusion of pulmonary vein. *C*, 3:19, after occlusion of pulmonary vein. *D*, 3:57, three minutes after occlusion of pulmonary artery, vein still occluded. *E*, 4:00, six minutes after occlusion of pulmonary artery, vein still occluded. *F*, 4:02, eight minutes after occlusion of pulmonary artery, vein still occluded. *G*, 4:13, nineteen minutes after occlusion of pulmonary artery, vein still occluded. *H*, 4:44, after 500 c.c. of systemic blood loss, with pulmonary artery and vein still occluded. Systemic blood pressure 80 mm. Hg, having dropped from an initial pressure of 120 mm. Hg. *I*, 4:47, after 700 c.c. blood loss, B P 50 mm. Hg. *J*, 4:58, blood pressure, 30 mm. Hg. *K*, 5:03, blood pressure, 25 mm. Hg.

In the three experiments wherein the lobar venous pressure was also recorded, this was found to parallel the changes in the lobar pulmonary artery pressure at a level lower by 5 to 10.0 mm. Hg.

B. *Effect of Occlusion of Pulmonary Artery and Vein of the Left Lower Lobe in Dogs With Expanded Bronchial Arterial System.*—

In the two dogs wherein an expanded bronchial arterial collateral system had been induced by previous ligation of the left pulmonary artery, the elevation in intralobar vascular pressure following occlusion of the left lower lobe artery and vein was more abrupt and reached a maximum in a shorter period of time than in the normal dog. (See Table I, Experiments Nos. 39 and 40). Whereas, the absolute peak pressure reached in Experiment 39 (45 mm. Hg) was not as impressive as that in Experiment 40 (75 mm. Hg) and actually fell short of that reached in four dogs with normal bronchial arterial systems, the

rise observed, in terms relative to the systemic blood pressure of 80 mm. Hg, is significantly higher than that in the dogs with normal bronchial arterial systems and normal systemic pressures (Figs. 6 and 7).

C. *Effect of Systemic Blood Loss and Retransfusion.*—

To study the effect of these factors, rapid blood loss was produced via a cannula in the femoral artery, the blood thus withdrawn being later returned under sufficient pressure to cause a rapid rise of blood pressure.

With the lobar pulmonary artery pressure already at a maximum due to occlusion of the lobar artery and vein, systemic arterial hypotension produced by hemorrhage caused a reduction of intrapulmonary blood pressure commensurate with the drop of systemic pressure (Figs. 5 and 8).

Restoration of the animal's peripheral blood pressure by rapid intra-arterial transfusion caused a return of the pressure in the occluded lobar pulmonary artery to its previous high level.

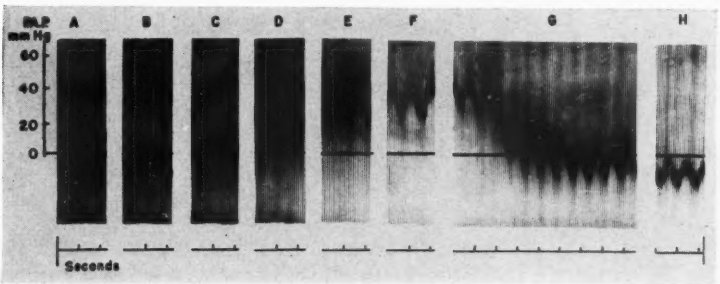


Fig. 6.—(Experiment 39.) Showing the rise in pulmonary artery pressure within the left lower lobe of a dog whose main left pulmonary artery had been ligated five weeks prior to the experiment. In this animal the systemic blood pressure was less than 80 mm. Hg throughout the course of the experiment, following the thoracotomy. The peak pulmonary artery pressures reached are high when considered in relation to the low systemic pressure. *A*, 1:10, base line. *B*, 1:17, one minute after occlusion of pulmonary artery. *C*, 1:20, immediately after occlusion of pulmonary vein, artery still occluded. *D*, 1:21. *E*, 1:22. *F*, 1:23. *G*, 1:24, during release of pulmonary vein. *H*, 1:27, after release of vein, pulmonary artery still occluded.

TABLE I. PRESSURE CHANGES FROM OCCLUSION OF PULMONARY ARTERY AND VEIN DISTAL TO OCCLUDED ARTERY

LOG NUMBER	PAP BASELINE (MM. HG)	AFTER PV OCCLUSION		AFTER PA AND PV OCCLUSION		SYSTEMIC BLOOD PRESSURE (SYSTOLIC MM. HG)
		MAXIMUM PAP (MM. HG)	TIME (MIN.)	MAXIMUM PAP (MM. HG)	TIME (MIN.)	
25	13	–	–	33	16	114
24	4	14	1	35	12	130
21	(5)	(15)	1	(10)	1	130
32	15	19	2	58	14	140
34	7	15	3	30	35	140
35	7	14	2	27	15	135
36	16	20	1	46	2	135
37	8	16	3	36	9	120
38	30	33	1	52	6	–
39	13	–	–	43	4	80
40	13	–	–	75	3	120
41	20	38	1	45	7	120
42	2	10	1	29	23	120

D. *Proof of Continual Influx of Bronchial Arterial Blood.—*

With the lobar pulmonary artery and vein occluded it was found that, if the pulmonary vein catheter cannulating the occluded lobar vessels be opened, blood would flow in a drip for an indefinite period of time from the end of the catheter. In order to rule out the possibility of passive outflow of blood from an engorged lobe, the external bleeding tip of the pulmonary vein catheter was elevated to a height of approximately 40 cm. above the pulmonary vein. Even against this imposed hydrostatic resistance, blood continued to drip at a rate of about six to eight drops per minute for prolonged periods.

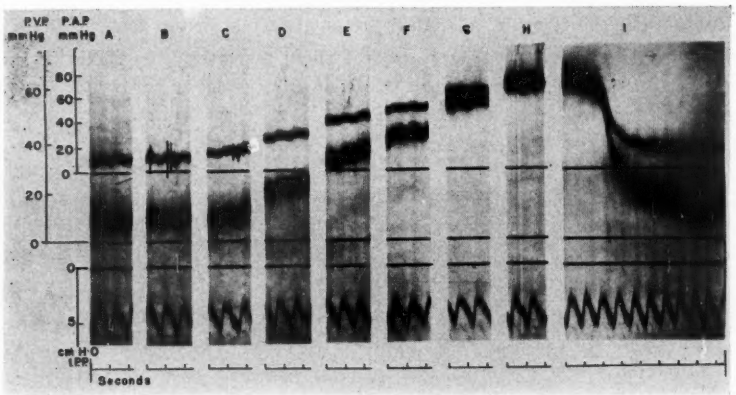


Fig. 7.—(Experiment 40.) Showing the rise in pulmonary artery and vein pressures of a dog whose main left pulmonary artery had been ligated six weeks prior to the experiment. In this experiment the effect of occlusion of the pulmonary artery alone is not shown, this vessel being occluded throughout the course. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural pressure (*I.P.P.*). *A*, 12:59, base line. *B*, 1:00, during occlusion of pulmonary vein. *C*, *D*, *E*, *F*, selections from a continuous run immediately after vein occlusion. *G*, one-half minute after occlusion of pulmonary vein. *H*, one minute after occlusion of pulmonary vein. *I*, 1:03, during release of pulmonary vein.

E. *Effect of Epinephrine.—*

With the lobar pulmonary artery pressure already at a maximum from occlusion of the lobar artery and vein, intravenous administration of epinephrine resulted in a still greater rise, occurring at the peak of systemic arterial pressure elevation, followed by a return to the pre-injection level paralleling the return of systemic pressure to normal.

F. *Other Effects of Occlusion of Pulmonary Artery and Vein of Left Lower Lobe.—*

I. *Systemic blood pressure:* There was no consistent effect upon systemic blood pressure due to occlusion of the vessels to a single lobe. In Experiment 30, which was one of the two dogs subjected to previous ligation of the left main pulmonary artery, there was a gradual decline of the systemic pressure over a twenty-five minute period following occlusion of both lobar vessels, from 100 mm. Hg to 50 mm. Hg. Upon release of the vessels there occurred a prompt return of the pressure to 80 mm. Hg. The fall might well be attributed to loss

of blood into the lobe, and the rise to auto-transfusion upon reopening of the connections with the systemic circulation through the pulmonary vein and left heart.

II. *Respiratory effects:* After lobar vascular occlusion changes in the spirogram were seen inconstantly. When present these consisted of a gradual decrease of the tidal volume, usually with acceleration of the rate after variable time periods. This might be the result of the reflex described by Churchill and Cope,[9] who noted that increased pressure within the vessels of a single lung when produced by rapid introduction of excessive amounts of blood caused rapid shallow respirations.

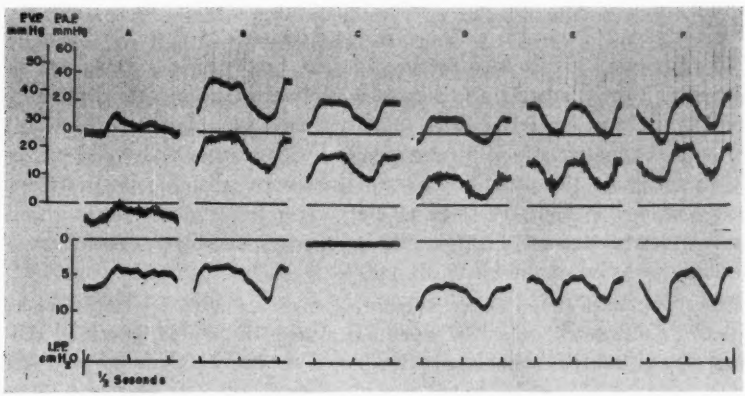


Fig. 8.—(Experiment 37.) Showing the effect upon intravascular pressure within the occluded lobe of systemic hypotension induced by hemorrhage and restoration of systemic pressure by retransfusion. Upper tracing—pulmonary artery pressure (*P.A.P.*); middle tracing—pulmonary vein pressure (*P.V.P.*); lower tracing—intrapleural artery and vein. *A*, 2:00, base line. *B*, 3:46, two minutes after occlusion of pulmonary artery and vein, blood pressure 120 mm. Hg. *C*, 3:55, after hemorrhage, blood pressure 75 mm. Hg. *D*, 4:00, after further hemorrhage, blood pressure 60 mm. Hg. *E*, 4:05, after retransfusion of blood, blood pressure 118 mm. Hg. *F*, 4:10, after further retransfusion, blood pressure 120 mm. Hg.

III. *Intrapleural pressure:* Pulmonary vascular congestion is known to produce changes in the dynamics of respiration which effectively alter the intrapleural pressure. Heyer and associates[10] induced congestion by administration of excessive amounts of fluid intravenously and found that there was increased amplitude of the intrapleural pressure swing with a consistent elevation of the expiratory pressure to values above atmospheric. In our experiments, with the congestion isolated to a single lobe, from occlusion of its particular artery and vein, consistent or significant effects on the intrapleural pressure were not seen.

IV. *Gross changes in the lobes:* The appearance of the left lower lobe varied depending on the period of time during which the vessels were allowed to remain occluded. Dense congestion was constant in some degree. If the period of occlusion was short, the color of the lobe was generally pink, whereas longer periods of congestion led to a dark, liver-like appearance, with the lobe enlarged and nonrepitant. Lung weight to body weight ratios were not computed. Gross evidence of pulmonary edema during the experiment, such as frothing of free fluid in the trachea, was not observed.

At the time of autopsy in each experiment the degree of occlusion of the vessels, whether by snare or balloon, was tested, and found to be complete in all instances, except in Experiment 21, where an apical segmental vein had not been included in the snare loop.

DISCUSSION

The rise in pressure observed within the pulmonary vascular bed of a lobe whose supplying pulmonary artery and vein had been obstructed is attributed to active congestion produced via the bronchial arteries. Although normally the bronchial arterial communications with the pulmonary circuit are by means of the intercapillary bed,[2] the intravascular pressures reached in the normal lobe after arterial and venous occlusion are far in excess of systemic (peripheral) capillary pressure. It is well recognized that the bronchial veins are poorly developed and are inadequate to accommodate more than one-third of the blood delivered by the bronchial arteries in unit time. This disproportion has been demonstrated anatomically and functionally by Berry and Daly[8] and by Bruner and Schmidt.[11] In the situation, then, of an obstructed pulmonary artery with impaired venous outflow, the so-called nutrient arteries of the lung become potentially a destructive force, raising the pulmonary vascular pressure far above the osmotic pressure of the blood plasma.

The phenomena noted above were in a sense predicted by Daly and his coworkers.[8] These workers, by an ingenious system of pumps, perfused the pulmonary artery and simultaneously the aorta or bronchial arteries of freshly sacrificed dogs, and then collected the blood from the left auricle and azygos vein. With pulmonary and aortic pressures both at normal limits, considerable blood passed from the bronchial to the pulmonary circuit. With the pulmonary artery pressure zero and aortic pressure normal, at least twice as much blood passed from the bronchial to the pulmonary circulation as from the bronchial circulation to the azygos vein. In the majority of their perfusion experiments an increase in aortic pressure raised the pulmonary arterial pressure as well as pulmonary venous flow.

Intravascular pressures observed in dogs with an abnormally "expanded" bronchial collateral circulation (produced by ligation of the left main pulmonary artery several weeks prior to the pressure studies) were elevated more rapidly and to proportionately higher levels following arterial and venous occlusion than in the normal dogs. The more rapid rise is to be related to expanded cross-sectional capacity and flow of the available bronchial arteries and a further development of extensive pre-capillary anastomoses between the bronchial and pulmonary arteries.[5] Thus the systemic pressure of the bronchial arteries is permitted to be more directly transmitted and felt in the pulmonary vascular bed. Increased bronchial collateral flow was demonstrated by our crude collection technique in the two dogs with previously ligated pulmonary arteries. An enormous increase in bronchial collateral flow following pulmonary artery ligation has been determined by the indirect Fick method and reported by Bloomer and associates.[12] It is unfortunately not known whether expansion of the

bronchial venous system parallels that of the bronchial arterial system after pulmonary artery ligation. If not, exaggerated rises would in part be due to a progressive discrepancy between bronchial arterial inflow and bronchial venous outflow.

The above-described pressure changes in occluded pulmonary arteries of normal dogs support the existence of functional anastomoses between the bronchial and pulmonary arteries, an assumption previously shown to be true in the isolated perfused dog lung by Daly and associates, and by others[6, 7] in various chronic human pulmonary disease states.

The physiologic effects of the expanded bronchial vascular system in pathologic states of the lung has been postulated by Liebow and associates to be as follows: (1) The high pressure effects of the expanded bronchial arterial system, anastomosing with pulmonary arteries at the pre-capillary level serve as a shunt or hydrodynamic block to the inflow of unoxygenated pulmonary arterial blood to diseased portions of the lung, thus helping to maintain a more nearly normal mixed arterial oxygen saturation in the efferent pulmonary veins. (2) Abnormal bronchopulmonary communications with their high flow rate, draining mainly to the left heart by way of the pulmonary veins, may play a significant role in the development of left heart failure. (3) Conversely, blood shunted into the pulmonary arteries by way of bronchial anastomotic channels tends to increase the pressure within the lesser circulation, thus increasing the work of the right heart, and predisposing to cor pulmonale.

The functional significance of the communications which have been demonstrated in the normal animals above is speculative at the present time. The bronchial arteries are richly supplied with autonomic fibers.[13] It is possible that variations take place in the flow through these vessels, constituting a mechanism whereby deoxygenated pulmonary artery blood is shunted away from poorly ventilated alveoli. This is merely an application to the normal animal of a theory advanced by Liebow and associates to explain the shunting of blood which occurs in various pathologic states. Further field for speculation lies in the possible role of bronchopulmonary anastomoses in the pathogenesis of pulmonary edema due to increased filtration pressures.

Of greatest interest and importance is the role these communications may play in the fate of a pulmonary segment or lobe which has been deprived of its direct pulmonary arterial blood supply by an embolus or other occlusive pathology. If such a segment had also a high degree of functional venous obstruction, as from chronic passive congestion or mitral stenosis,[14] it is not improbable that bronchial collateral inflow at systemic pressures would produce a destructive imbalance, tending to favor the development of capillary engorgement, increased filtration, edema of the alveoli and interstitial tissues, diapedesis, and frank hemorrhage. This may be the mechanism of hemorrhagic infarction.

SUMMARY

When the pulmonary artery and vein of a lobe in the dog's lung are both occluded in an otherwise intact chest, the vascular pressures within the lobe

rise to abnormally high levels. This in vivo evidence supports the existence of functional communications between the bronchial and pulmonary arteries within the normal lung.

The possible significance of these communications is discussed.

We are indebted to Professor Averill A. Liebow, Department of Pathology, Yale University School of Medicine, for many valuable suggestions, and Mr. Armand Negri, Department of Surgery, Yale School of Medicine, for his technical assistance.

REFERENCES

1. Miller, W. S.: The Lung, Springfield, Ill., and Baltimore, 1937, Charles C Thomas, pp. 209.
2. Miller, W. S.: The Arrangement of the Bronchial Blood Vessels, Anat. Anz. **28:** 432, 1906.
3. Ghoreyeb, A. A., and Karsner, H. T.: A Study of the Relation of Pulmonary and Bronchial Circulation, J. Exper. Med. **18:** 500, 1913.
4. Berry, J. L., Brailsford, J. F., and Daly, I. deBurgh: The Bronchial Vascular System in the Dog, Proc. Roy. Soc. Londcn, 109B: 214, 1931-32.
5. Liebow, A. A., Hales, M. R., Bloomer, W. E., Harrison, William, and Lindskog, G. E.: Studies on the Lung After Ligation of the Pulmonary Artery. II. Anatomical Changes, Am. J. Path. **26:** 177, 1950.
6. Wood, D. A., and Miller, M.: The Role of the Dual Pulmonary Circulation in Various Pathologic Conditions of the Lungs, J. THORACIC SURG. **7:** 649, 1937-38.
7. Liebow, A. A., Hales, M. R., and Lindskog, G. E.: Englargement of the Bronchial Arteries and Their Anastomoses With the Pulmonary Arteries in Bronchiectasis, Am. J. Path. **25:** 211, 1949.
8. Berry, J. L., and Daly, I. DeBurgh: The Relation Between the Pulmonary and Bronchial Vascular Systems, Proc. Roy. Soc. London, 109B: 319, 1931-32.
9. Churchill, E., and Cope, O.: Rapid Shallow Breathing From Pulmonary Congestion and Edema, J. Exper. Med. **49:** 531, 1929.
10. Heyer, H., Holman, J., and Shires, G.: The Diminished Efficiency and Altered Dynamics of Respiration in Experimental Pulmonary Congestion, Am. Heart J. **35:** 463, 1948.
11. Brunner, H. D., and Schmidt, C. F.: Blood Flow in the Bronchial Artery of the Anesthetized Dog, Am. J. Physiol. **148:** 648, 1947.
12. Bloomer, W. E., Harrison, W., Lindskog, G. E., and Liebow, A. A.: Respiratory Function and Blood Flow in the Bronchial Artery After Ligation of the Pulmonary Artery, Am. J. Physiol. **157:** 317, 1949.
13. Larsell, O.: Aspects of Innervation of Lung, Northwest Med. **22:** 311, 1923.
14. Dexter, L., et al.: Studies of the Pulmonary Circulation in Man at Rest. Normal Variations and the Interrelations Between Increased Pulmonary Blood Flow, Elevated Pulmonary Arterial Pressure, and High Pulmonary "Capillary" Pressure, J. Clin. Investigation **29:** 602, 1950.

Erratum

In the paper by Winfield O. Kelley, M.D., and David V. Pecora, M.D., Norwich, Conn., entitled "Revision Thoracoplasty" (J. THORACIC SURG. **22:** 295, 1951), on page 295 in the first paragraph on line 19, the sentence "This method was successful in converting the sputum of 4.35 per cent of twenty-three cases" should read "This method was unsuccessful in converting the sputum of 4.35 per cent of twenty-three cases."